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The Antiseptic

Estd. 1904

A Monthly Journal of Medicine & Surgery

Published on the 15th of every Month

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Asst. Editor : U. VASUDEVA RAU, M.B., B.S.

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Original Articles

CONCEPT OF MYOCARDIAL INSUFFICIENCY AND THE MODERN TRENDS IN ITS TREATMENT*

PART I.

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MOST of the clinical conditions end sooner or later in circulatory or cardiac failure. It is, therefore, a condition commonly encountered in medical practice. The nature and the treatment of cardiac failure has continued to attract the attention of the clinicians and the experimental investigators for many years past. The fundamental step which put the phenomenon of heart failure on a sound experimental basis was taken by the work of Starling, Wigger, Katz and others. With his heart-lung preparation Starling established the Law of Heart so fundamental for the clinical study of cardiac failure. It enunciated that 'within physiological limits, the larger the volume of the heart, the greater the energy of contraction.' Thus dilatation of the heart is not solely a manifestation of impaired contractility and in itself purely deleterious to the function of the myocardium, but may at times be a useful compensatory process serving to increase the capacity for work of the dilated chambers. For this reason Rosenbach introduced the term 'Hyper-diastole', meaning thereby the supernormal filling of the cardiac chamber during diastole.

* Specially contributed to THE ANTISEPTIC.

Since then the progress in the investigation of myocardial insufficiency has been rapid. Quantitative methods have been introduced for the measurement of some of the fundamental circulatory variables in health and disease. The estimation of cardiac output, previously hampered by clinical difficulties, has been achieved by means of cardiac catheterization. In 1929, Forssman passed a ureteric catheter through his own median basilic vein, into his right auricle. He then walked to the X-ray department and had it photographed *in situ*. In 1932, radio-opaque material was passed through the catheter into the heart by Monis and Carvalho, in Spain, and pictures of the pulmonary vessels were obtained. In 1941, Cournand and Ranges left the catheter in position for an hour, while making successive observations, clotting being prevented by running saline slowly and continuously. This new method led to important advances in three directions :—

(1) It has enabled the measurement of pressures in the right auricle and ventricle directly.

(2) It has enabled the accurate estimation of the cardiac output.

(3) It has advanced angio-cardiography by introducing radio-opaque material directly in heart.

Here mention should be made of the introduction of Ballisto-cardiograph by Starr in 1941, for the purpose of the estimation of the cardiac output. It measures the recoil of cardiac contraction. The principle is that the blood ejected from the heart in systole makes the body recoil towards the feet, as it moves towards the head. Then as the blood column moves down along the descending aorta, the recoil is in the opposite direction. These movements are transmitted to a suspended table on which the patient is lying, and are photographed. The size and shape of the deflection obtained enable the output to be estimated. By this method cardiac output can be measured during relaxed condition of the patient, and vascular punctures and auricular catheterization are obviated.

A method of investigating the volume of the heart was devised by Nylin, in Stockholm, in 1943. Two simultaneous skiagrams are taken at right angles to each other. Transverse and A.P. diameters of the heart can then be measured. From these findings, accurate volume of the chambers can be calculated.

Prinzmetal, in 1949, introduced Radio-cardio-cardiography to measure the emptying time of the heart. Radio-active Sodium is injected intravenously and the concentration of the particles is obtained by a Geiger-Muller counter and recorded on a revolving drum. The method is useful in three ways :—(1) It estimates delay from the chambers ; (2) It measures the emptying time ; and (3) It indicates filling pressure separately for each chamber. This

method is expected to be useful in adding to our knowledge of hæmo-dynamics in cardiac failure.

John Hutchinson introduced 'Vital Capacity' Test in 1846, for use in pulmonary diseases. Peabody, in 1921, applied the method to the problem of cardiac dyspnœa. The test is quick and easy as you all know. A young athlete may puff in 5000 c.c. while the patient with cardiac failure barely manages to put in 1000 c.c. The prolonged ending by squeezing in the last few hundreds has proved to be a functional test of emphysema, and in lower ranges is seen also in patients with cardiac failure following emphysema.

In 1926, Blumgart and his associates injected a radio-active substance into the basilic vein and were able to detect its arrival at the brachial artery of the other arm. Thus, they were able to measure the time taken by the blood to flow up the vein to the heart, through the pulmonary circuit to the left ventricle, and finally out in the brachial artery. Many other simpler but less accurate methods have been used to the same end, *viz.*, to estimate the circulation rate. Endpoint in the different tests is different, *viz.*, taste in Decholin, Mag. Sulph and Saccharin tests, flushing of the skin in Histamine test and Amyl Nitrite test, odour of breath in Ether test, etc. Apprehension, thyrotoxicosis, anæmia, emphysema increase the rate. These are the conditions in which the cardiac output is high. The rate is slow in myxœdema, polycythæmia, and congestive cardiac failure. In cardiac failure the slowing may be about thrice the normal time.

These modern methods of investigation have tended to remodel our views on cardiac failure. Using these methods McMichael and Sharpey-Schafer have been able to show that cardiac failure may be present with output still above the normal. They also showed that the cases of cardiac failure, with high output, have a fast circulation rate, while those with lowered output have a slow circulation rate.

Critical consideration of newer concepts is not only desirable but also actually necessary for the scientific handling of patients. Some take the extreme view that the congestive heart disease and circulatory failure result primarily from renal changes and disorders of salt and water mechanism. It must be admitted that the œdema is the most important manifestation of myocardial failure and dissipation of œdema is accomplished by increasing the output of Sodium in urine. But the fact remains that the myocardial insufficiency begins in the heart muscle. The generally accepted conceptions of the functional origin of the symptoms and signs that accompany the weakening of the heart muscle support mainly the back pressure failure. But the evidence of forward failure in some conditions is definite. The blood flow through renal glomeruli in either condition is inadequate and Sodium is retained, and œdema appears. Thus, some revision of our old concepts is necessary in the light of modern

methods. A broader and more comprehensive concept of genesis of signs and symptoms of myocardial insufficiency must be evolved which may modify the treatment as well.

Mechanical work accomplished by the ventricle depends upon its fibre length. Starling has pointed out that oxygen consumption increases with fibre length. As long as oxygen supply keeps pace, the strength of the ventricular contraction and the cardiac output will respond to increasing dilatation. Circulatory equilibrium will be maintained, upto the critical diastolic volume and muscle stretch, beyond which oxygenation lags and ventricles fail to empty themselves. Inadequacy of the oxygen supply to the myocardium, especially the amount and availability of extra oxygen necessary for the accomplishment of extra mechanical work, will cause myocardial efficiency to drop off, and a relative insufficiency in the cardiac output develops. The ventricular cavities later dilate with an absolute decrease in the cardiac output. The classic picture of congestive or backward heart failure develops as the gradient of blood flow between the pulmonary and the aortic systems drops off, and as the blood accumulates in the venous channels. This backward failure appears early in rheumatic carditis and coronary insufficiency, while there is still considerable myocardial reserve[1].

Circulatory or forward failure may develop in several conditions, in which the arterial bed is widely dilated, as in aortic regurgitation, severe anæmia, hyperthyroidism and beri beri. In these conditions, during failure, the cardiac output has been shown to be normal or actually increased, but still inadequate. The minute volume circulation is normal or above, yet the blood flow, actually provided to the tissues in the widely dilated bed, may still be below the critical minimum required for the body as a whole.

As a result of this new orientation of the views, two types of cardiac failure can be visualized, *viz.*, one with diminished or low cardiac output and low pulse pressure, resulting in backward or congestive failure, and the other with normal or supernormal but yet inadequate ventricular output, and high pulse pressure resulting in forward or circulatory failure[2]. In both types acute and chronic forms may be present. The acute backward or congestive failure develops as a result of acute insufficiency or left ventricular failure with extreme dyspnœa or orthopnœa and acute pulmonary œdema. The chronic backward failure may arise in myocarditis, myocardial affections due to gradual coronary sclerosis, diastolic hypertension, hypothyroidism, chronic pericarditis etc. Here, besides lung congestion, there are signs of right ventricular failure with engorgement of liver, ascitis and dropsy in dependent parts.

Acute cardiac failure occurs in cardiac standstill, shock, hæmorrhage, extreme tachycardia or bradycardia. Chronic forward or circulatory failure, with high systolic output and high pulse pressure, yet insufficient minute volume circulation occurs in aortic

regurgitation, systolic hypertension, hyperthyroidism, arterio-venous shunt, congenital lesions, anæmia and beri beri.

Clinical manifestations which are considered evidence of myocardial insufficiency have their origin in the failure of heart muscle in ventricular contraction, to supply the tissues with blood adequately. At a certain critical stage heart is not able to increase the ventricular output relatively or absolutely in response to the demands of adequate circulation. Cardiac output then falls below the level necessary for adequate renal function and oxygenation of tissues. Now the clinical picture of the 'high output failure' and 'low output failure' is almost identical, irrespective of the fact whether the inadequate output is relative or absolute.

When the myocardium begins to fail, the following important events take place :—

(1) The cardiac output falls relatively or absolutely, according to whether it is 'high' or 'low' output failure.

(2) There is retention of sodium and water in the extracellular tissues due to inadequate renal circulation.

(3) The venous pressure rises. This can be attributed to: (*a*) reflex veno-constriction as claimed by McMichael and Sharpey-Schafer; (*b*) increase in the blood volume; and (*c*) weakness of myocardium.

(4) The blood volume increases, due to hæmodilution, resulting from retention of salts by the kidneys.

(5) The circulation rate slows.

(6) Starling noted constriction of the arteries with dilatation of the arterioles and pooling of the blood in the veins. Extra blood is also contained by the dilated chambers of the heart. Both these facts necessitate an increase in the blood volume.

The sequence of events is by no means continuous, and is complicated by the effects of rest and treatment. (Treatment will be discussed in a subsequent paper).

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"Jayashree,"
Deshmukhwadi,
Poona City.

SEDATIVES IN HEART FAILURE*

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THE patients with heart failure are often seen in a very anxious state. Part of the anxiety is psychogenic, part is due to repeatedly disturbed nights, and part, of course, is due to the failing circulation.

A right sedative given at the right time may make all the difference in the prognosis. More often than not, the right sedative is Morphine. But other sedatives have their proper place. The various sedatives described in this article had been administered to some 30 patients with heart failure. Majority of the patients were in right and left ventricular failure. At least one patient had only left ventricular failure following hypertension. It is not meant to belittle the importance of other standard measures such as Digitalis, Mercurials, etc., which in a way do help to sedate the patient.

Morphine.—This drug relieves the attack of cardiac dyspnœa like a miracle. The dyspnœa is due to the abnormal stimulation of the respiratory centre and disturbance in the reflex respiratory regulation. The reflexes originate in the lungs and the chemoreceptors in the carotid-sinus. Pulmonary congestion produces reflex hyperventilation. This may exhaust the patient by increasing his burden. Morphine helps by diminishing the excitability of the centres. Morphine relieves dyspnœa through a depression of the central sensory perception. All of us who use Morphine rationally, have seen the gratifying results obtained in patients who have spent sleepless nights and restless days. These unfortunate people suffered just because Morphine was withheld from them. I vividly remember a patient in severe dyspnœa, who had not known what is sleep or rest for six days and nights, falling immediately to sleep after a deep intramuscular injection of 1/6 gr. Morphine. Next day he was a different man. He was extremely grateful for the rest he had. I felt that if there is any joy in the practice of medicine and cardiology it is this :—

Morphine is described as "Second Digitalis." I feel that it is the "First Digitalis", because it gives prompt relief during the period taken by Digitalis to have a hold on heart failure. At this juncture, the contra-indication to Morphine must be stressed. In any pulmonary dyspnœa and in 'Cor Pulmonale' it is absolutely contra-indicated. Morphine does have anti-diuretic effect, but this objection should not weigh too much when subjective relief from dyspnœa is to be obtained.

Clinical experience as early as 1850 has conclusively shown that Morphine has no deleterious effects in heart failure and the deleterious effects sometimes observed were due to the gravity of the disease rather than to Morphine. My plea is, too much of time should not be spent in trials of Barbiturates, Chloral and Bromides when the vicious circle can be stopped by Morphine.

* Specially contributed to THE ANTISEPTIC.

When psychosis sometimes accompanies severe heart failure, Morphine and Barbiturates may be harmful. In such a condition Paraldehyde may be the drug of choice.

Dosage of Morphine:—$^1/_6$ gr. to $^1/_4$ gr. hypodermically once or twice in a day till the action of other therapeutic agents is established.

Codeine.—It is generally used for control of cough when cough is a very distressing feature, especially, in some hypertensive failures, it works wonders. Cough is described as equivalent of cardiac asthma. Codeine is only one fourth as potent as Morphine in its ability to cause subjective and respiratory depression. Sleep induced by Codeine is not so refreshing as by Morphine. Hence it cannot be relied upon solely in allaying distress in severe heart failure. In high dosage, it may cause excitement. In two patients, one with hypertensive failure and the other with failure due to mitral stenosis, the cough was so severe that it was the major element in apprehension. One of the patients was given three and half grains of Codeine in 24 hours with most gratifying results. The pulmonary congestion is the main cause of cough. Bouts of cough tend to increase the venous return to the right side of the heart and this overburdens the pulmonary circuit. Hence the relief after Codeine is obvious.

Pethidine (Demerol):—It does not depress the respiratory centre so much as Morphine does. But it is not as effective as Morphine. Its property of relieving smooth muscle spasm is an additional advantage which might be exploited in certain cases of cardiac failure with broncho-spasm. It does not excite vomiting. Hence it is particularly useful when Morphine produces vomiting. It is not superior to Morphine, and it does lead to addiction. Oral dose is not always effective. Intramuscular or subcutaneous dosage of 50–100 mgm. is commonly employed. In my experience the drug is quite satisfactory, though I have never taken chance with it, in severe dyspnœa, which was not attended to for a long time. For such cases, I have always depended on Morphine.

One unique indication for Demerol, as contrasted to Morphine, is in dyspnœa of pulmonary origin and in Cor Pulmonale. Barach recommends Demerol in these conditions. I have no personal experience with this drug in Cor Purmonale. In fact, I have never been bold enough to give it in this variety of heart failure, knowing the dangers of Morphine in Cor Pulmonale. Clinical trials of Demerol in Cor Pulmonale will be awaited with interest, because in this type of heart failure the physician is helpless as Digitalis does not help and is actually contra-indicated. I have given Demerol intramuscularly, to a patient with hypertensive failure, for a long time, with gratifying results.

Papaverine.—It is rarely used alone for its sedative action. Its coronary dilator action may be a quality worth exploiting. Some continental authorities have used it frequently as a sedative

in heart failures. But the consensus of opinion is not in favour of relying on it as opposed to Morphine. Dosage is 30–80 mgm. intramuscularly or orally and 10–60 mgm. intravenously. Attempts are made by one manufacturer to combine Papaverine and Codeine in a single injection. Its usefulness in cardiac failures remains to be proved.

Barbiturates.—These have depressed action on the cerebrospinal axis. In heart failure they are used mainly for their hypnotic effect. Sometimes, they are useful in allaying apprehension in a very sensitive patient. But they can never stand any comparison with Morphine. At this stage I should like to stress that in a patien twith severe dyspnœa and restlessness no Barbiturate should be given instead of Morphine. Barbiturates are better given at night as hypnotics when circulation has improved after 2–3 days with Morphine and Digitalis.

For hypnosis Diallylbarbituric Acid (Dial) or Seconal or Amytal or a combination of Seconal and Amytal (Tuinal) are useful. I have found these agents useful in 15 cases.

For sedation, Phenobarbitone is the drug of choice. It is given in ¼–½ gr. doses twice or thrice a day.

Chloral Hydrate.—It is one of the cheapest and most effective hypnotics. In dosage of 15–20 grs. it produces sleep. It is indicated when the patient with heart failure is not getting sleep because of nervousness. The same remarks apply to this drug as have been made for Babiturates. It can also be given rectally in 5 to 6 ounces of water.

Bromides.—The same general remarks apply to this drug as for Barbiturates. A traditional combination of Chloral and Bromides is commonly used. It should be remembered that Bromides take two days before their action is evident. This is because, adequate concentration of the iron in extracellular fluids has to be reached. When rapid hypnosis is required, other hypnotics should be used. A single dose of Bromides can have no effect.

Paraldehyde.—It is a safe and effective hypnotic having the same place in the therapy of heart failure as the Barbiturates and Chloral. Its nauseating smell is the drawback. It may be given rectally 10–20 c.c. in two parts of Olive oil. It can be given intramuscularly 4–8 c.c.

In this article, even at the risk of some repetition, I have tried to put the various sedatives, in their proper place, in the management of heart failure. If used with proper understanding of the pathogenesis and pathologic physiology, these measures help to alleviate the sufferings of the patients.

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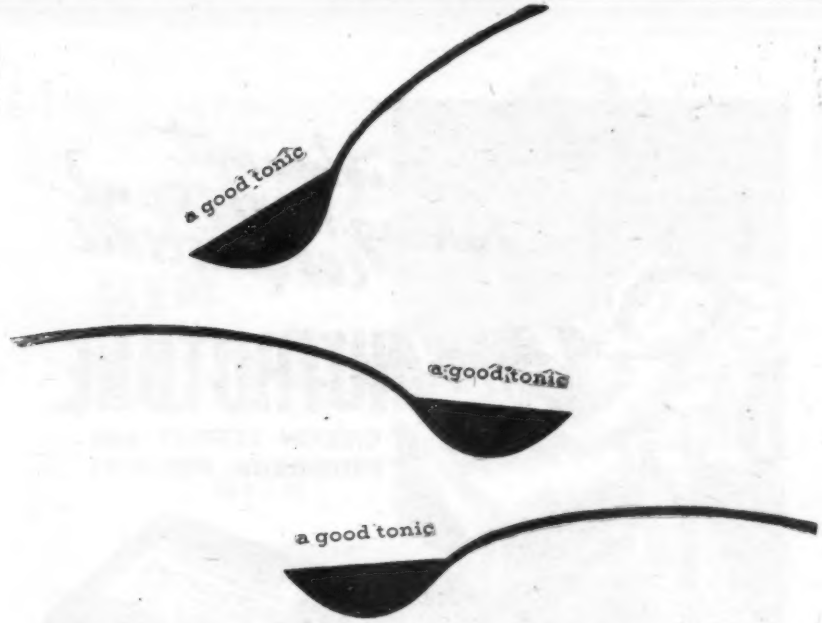

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AUREOMYCIN—
AN EVALUATION OF ITS PRESENT STATUS*

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THE subject for today's discussion 'Aureomycin' will be reviewed in the light of reports published in the medical press thus far. As it is, we cannot judge the merits or demerits of this powerful antibiotic in so short a time as two years within its discovery. Anyhow an attempt will be made, in this short paper, to present in a consolidated form, abstracts from the comprehensive literature that is available. Unfortunately, few reports have been published in this country and these too concern chiefly the typhoid group of fevers.

The year 1948 opened up a new chapter in the history of antibiotics with the announcement of the discovery of Aureomycin, by Dr. B. M. Duggar, of the Lederle Division, of the American Cyanimid Co. in New York. This antibiotic has been described by earlier observers as 'the most versatile of the antimicrobial drugs in present day use, with Chloromycetin a close second and with Terramycin, Polymixin and Bacitracin still running. Of course, time, experience and end results alone will show which of these, provided nothing new is discovered in the meanwhile, wins the race to subdue the widest range of bacterial activity with a minimum of toxicity and at a low cost. If medical research continues at the speed at which it has been working since the last fifteen years, it would not be inconceivable to dream of a drug having capabilities of destroying the malarial parasites, the cholera vibrio, Bact. typhosus, small-pox virus, End. histolytica, and lethal to the lepra and the tubercle bacillus, all in one pill; this would be indeed a dream fulfilled for many of us, who are already slowly but surely giving up the use of their clinical judgement and knowledge and are indulging in haphazard treatments. It is not a very rare occasion when we come across a patient who has been treated on the following lines for his fever: At the outset he is put on anti-malarial drugs and Salicylates; a few days later he is switched on to one of the compounds of the Sulpha group; thereafter he is advised a course of Penicillin injections and/or Emetine; if within a few days he has not improved or given up treatment he is given Streptomycin and Vitamin preparations; and if he unfortunately still survives financially, physically and mentally, he is sincerely advised to give a trial to Para-aminosalicylic-acid, or even Chloromycetin. Thanks to the prohibitive price of Aureomycin its reputation has not suffered like its companions in the field. I am sure if a patient like the one referred to above is examined a bit more carefully, history elicited more diligently along with the necessary

* Paper read before a meeting of the Clinical Association, Civil Hospital, Mandi.

laboratory and radiological aid, then a single chemotherapeutic agent or a judicious combination of any of these anti-microbial drugs that are at our disposal today, would have gone a long way in alleviating the suffering and preventing what I would term as 'a discouraging waste of time, money and energy.' Let us therefore be not led away by the huge array of antibiotics that we, by virtue of this noble profession, are privileged to use for the betterment of suffering humanity. The greater the number of such drugs the greater the need for an accurate diagnosis and hence a healthier prognosis, by the selection of the aptest therapy. Along with the knowledge of what these antibiotics will do, we must also bear in mind what they are unlikely to do, in order to avoid needless expense to the poor patient and disappointment to the physician. Homœopathic dosage of any antibiotic, as a rule, will do more harm than good. Hence adequate dosage of the specific therapy should be given a fair trial before it is condemned. Failures in treatment will then be far less than what they are today. Advantage should also be taken of the synergistic action of such drugs. "It should be remembered that Sulphonamide drugs, Penicillin, Streptomycin, Aureomycin and Chloromycetin, all attack and damage different systems in susceptible infecting micro-organisms. The intelligent exploitation of this knowledge will result in prompt and satisfactory cures of infectious diseases."[1]

Let us now see what Aureomycin promises us before we are bejewelled by this gold-coloured mould.

Pharmacology.—Aureomycin [also known as Duomycin] is a gold-coloured (crystalline hydrochloride) antibiotic, prepared from Streptomyces Aureofasciens, and which deteriorates rapidly in alkaline solution at room temperature.

Action.—The action of Aureomycin, according to Chandler and Bliss[2], is bacteriostatic rather than bactericidal. According to Finland *et al*[3] most organisms do not become resistant to it as they do to Streptomycin or Penicillin. Price and his co-workers[4] have demonstrated the existence of a synergistic relation between Aureomycin and Polymixin or Penicillin *in vitro*, and between Aureomycin and Penicillin *in vivo*.

Aureomycin, considered the most versatile of the antibiotics, has a range of activity overlapping that of both Penicillin and Streptomycin and extending into regions untouched by either. It has been found effective not only against a wide variety of gram-negative and gram-positive bacteria, many of the strains resistant to the other antibiotics, but against also those classes of very minute micro-organisms against which no effective weapon had previously been available. Its ability to traverse the meningeal barrier and appear in therapeutic amounts in the C. S. F. adds to its value in meningitis caused by susceptible organisms.

Aureomycin has been shown to be potent against all rickettsial, many bacterial, certain viral, and some parasitic infections. Before I go further into the details of the antibiotic potentialities of this drug it would be interesting to throw light on another function of Aureomycin as reported in the lay press[5]. In this short note is mentioned the growth-stimulating properties of Aureomycin. "According to the scientists of the Lederle Laboratories (Pearl River, New York), the addition of minute quantities of powdered Aueromycin to hog and poultry feed accelerated the growth of test animals by as much as 50%. Such a growth response "cannot be duplicated by any of the vitamins known at the present time, even when they are given in many times their normal requirements." Early medical reports on the successful use of Aureomycin in treating infectious diseases of children showed that the drug also increased the appetite, generally improved health, and sometimes stimulated growth. Doctors in many U.S.A. hospitals are now investigating the potentialities of Aureomycin in stimulating the growth of malnourished and undersized children. The vitamin-like qualities of Aureomycin were discovered by accident while Lederle Biochemists were studying Vitamin B_{12}, the anti-anæmia vitamin. Vitamin B_{12}, also a growth stimulator, is extracted from the fermentation residues left after production of antibiotics like Streptomycin, Penicillin and Aureomycin from soil moulds. The Vitamin B_{12} extracted from the Aureomycin residue seemed to have more potent growth effects than those derived from other sources. The scientists after further research found the extra growth factor to be Aureomycin itself.

Requisite blood levels of Aureomycin.—Various workers have made attempts to determine the dosage of this drug to various diseases and consequently engaged themselves in obtaining blood levels after the institution of Aureomycin therapy in human beings and in laboratory animals; their results lead one to conclude that a dosage of 0·5 to 1·0 gm. is sufficient to maintain an effective blood level for about six hours. Dr. Perrin H. Long[6], Baltimore, replying to a question during a discussion at the annual meeting of the American Medical Association in 1949, has very aptly cleared the position regarding blood level estimations of Aureomycin. He says: "I do not recommend determining Aureomycin blood levels as a routine procedure. I believe that if one sees the patient a couple of times a day and watches his temperature chart and his clinical condition one knows whether he has had enough Aureomycin; if his temperature is not down to normal in 48 hours and you are treating him for something that you know can be cured by the drug, then give the patient a little more Aureomycin."

Administration, absorption and excretion.—Herrel and Heilman[7] have demonstrated that Aureomycin is readily absorbed into the general circulation and quickly distributed in many body fluids and tissues. It is capable of diffusing through the meninges,

the pleura, and the placenta, and is excreted into the bile as well as into the urine. The oral administration of 0·75 to 1·0 gm. promptly produces therapeutic serum levels (averaging 2–4 micrograms per c.c.) which are maintained for a number of hours and measurable activity may persist for as long as 30 hours. Lepper and his colleagues[8] were able to detect Aureomycin in the cerebrospinal fluid in 6 out of 9 of the patients, the concentration seeming to depend on the height and duration of the blood levels. In one patient the breast milk was assayed and no Aureomycin was found, even with high blood concentrations. In all cases, the drug appeared rapidly and in high concentration in the urine.

Harned, Cunningham and co-workers[9] found that following oral administration of Aureomycin Hydrochloride, the rate of excretion remained relatively constant for at least 6 hours, while after intravenous injection the excretion was rapid during the first two hours, slower between the second and fourth hours, and low between the fourth and sixth hours. Aureomycin was essentially without effect on the blood sugar, or on the activity of the isolated gut or uterus of guinea pigs or rabbits. No evidence of antipyretic or antihistaminic action was noted. It was found that Aureomycin was able to pass the blood brain barrier into the C.S.F. in therapeutic amounts, but no qualitative measurements were made. It appeared to be without effect on the central nervous system. No evidence of methæmoglobin formation was observed following intravenous dosage. Careful tests have shown no evidence of urinary impairment, jaundice, or changes in icteric index. Cephalin flocculation and prothrombin have been found normal. Repeated blood examinations have not indicated the development of anæmia or of any hæmolytic process, leucopenia or thrombopenia. Blood chemistry tests have shown normal values for total protein, albumin globulin content or ratio, cholesterol carbon dioxide, and alkaline phosphatase values. Aureomycin Lederle, is relatively non-toxic, but patients may become allergic to it in the same way that they become allergic to any other antibiotic. With the improvement in manufacturing processes and the elimination of impurities the side effects noticed following its use may also disappear.

On account of the nausea and vomiting that occurred following its use in some patients, earlier workers advised the use of Aluminium Hydroxide to combat this reaction, but recently Di Gangi, F. E. and Rogers, C. H.[10] found that this alkali had a high absorption affinity for Aureomycin thus explaining the low blood levels of this antibiotic that have been observed clinically when Aluminium Hydroxide Gel was administered simultaneously. These workers treated several solutions of Aureomycin Hydrochloride with this Gel and after two hours assayed for potency both colorimetrically and bacteriologically, and they found that there was almost complete removal of Aureomycin from the solutions.

Waisbren *et al*[11] reporting their observations confirmed (*in vivo*) the views of Di Gangi *et al*. They observed 11 human subjects prior to and after they were given Aluminium Hydroxide Gel. Ten of their subjects showed a decided drop in Aureomycin serum levels within 24 hours after they were given two tablespoonfuls of this Antacid with each 0·5 gm. dose of Aureomycin, thus proving that Aluminium Hydroxide Gel interferes with absorption of Aureomycin in the gut and may in certain infections lower the amount of Aureomycin in the serum and tissues below an effective blood level. Therefore Aluminium Hydroxide Gel must not be used along with Aureomycin.

Long[6] suggests the use of Vitamins in amounts adequate to supply the total daily requirements of the accessory food factors to patients receiving Aureomycin for extended periods. His suggestion is based on the possibility that the disturbances of intestinal function often noted in these patients may produce subclinical vitamin deficiencies.

Toxic reactions.—Until recently the only side effects encountered in patients on Aureomycin therapy were :—Nausea, vomiting, anorexia, loose odourless bowel movements not amounting to a frank diarrhœa, due perhaps to its bacteriostatic action on the intestinal bacteria; excoriations around the anus; epigastric distress. These reactions did not prohibit the continuance of its use. Reactions following intramuscular injections were local pain and irritation at the site of the injection. No local effects were observed during intravenous therapy, or during instillations into the eye of the Borate (0·5%) solution.

Recently reports have appeared in the medical press pertaining to further undesirable reactions. Harris *et al*[12] report rapidly developing mucous membrane changes in many female patients, attributed to Vitamin B deficiency through destruction of intestinal bacteria. Peck and Feldman[13] have reported three cases of allergic skin eruptions due to Aureomycin. One eruption was urticarial in type, the second presented an erythema-multiforme-like eruption and urticaria, the third was an eczematoid eruption in the groin and on the scrotum. Scratch and intradermal tests as well as patch tests with Aureomycin failed to elicit a positive reaction. Re-elicitation of the eruptions in each instance on the administration of Aureomycin was evidence that the patients were sensitive to this drug. Apparently the skin eruptions which occur after the administration of Aureomycin follow the pattern of most drug eruptions in that they nearly always are associated with negative skin tests to the responsible drug.

Parets[14] describes a severe reaction consisting of angioneurotic œdema of the face and a fairly generalised nonpruritic erythematous skin eruption occurring on the seventh day of treatment with Aureomycin in doses of 2 gm. daily for early bronchiectasis. This reaction lasted for a week. Treatment with the various anti-histaminic

drugs was of no benefit. This patient was unfortunately sensitive to a number of drugs administered to him at various times. Quoting other workers on the subject, Parets, in the same article, describes Spink *et al*[15] and Brande *et al*[16], an evanescent type of reaction consisting of an abrupt rise and fall in temperature occasionally accompanied with a shock-like picture with a drop in blood pressure and tachycardia during the course of treatment of brucellosis. This is believed to be akin to the Herxeimer reaction. Rodriquez *et al*[17] have observed a true febrile Herxeimer response frequently during treatment of early syphilis when large doses of Aureomycin are administered, but a cutaneous Herxeimer reaction has not yet been observed. Lennette *et al*[18] noticed an inflammation of the mouth and scrotum as well as pruritic papular lesions on the shoulders of patients during the course of Q fever therapy. These symptoms did not necessitate the cessation of therapy or become progressively worse while administration of the drug was continued.

An interesting case of a severe reaction to Aureomycin has been recently reported by Riese[19]. As an experimental treatment for obstructive jaundice thought to be due to a virus infection he administered Aureomycin to a patient at the rate of 3 gm. daily for six weeks. At the end of this period " burning in the chest " and swelling of the legs appeared, but the symptoms subsided within two days after the patient discontinued the treatment. Aureomycin was re-administered and at the end of two weeks a toxic reaction, consisting of œdema of the face, hands, forearms, ankles and feet developed, in association with a papulovesicular eruption beneath the breasts, between the thighs and around the anus. The patient had burning sensations in the mouth, where an inflammatory reaction was noted. Symptoms again disappeared after the withdrawal of the drug within a few days. Later as little as 1·25 gm. of Aureomycin caused a reappearance of the symptoms indicating that true sensitisation had developed.

Although a number of toxic reactions have been enumerated above during therapy with Aureomycin Hydrochloride, it is at once apparent that the discontinuation of this antibiotic promptly relieves the distress caused by it and within a few days all these toxic phenomena disappear.

The clinical use of Aureomycin.—Ever since the historic announcement of Aureomycin by Duggar, endless reports have appeared in the medical press all over the globe attributing hitherto unexcelled potentialities to it. It is being claimed to have cured more than half of the total diseases met with, and, with the vast literature still piling up, very shortly, it would be claimed to be a drug par excellence, a panacea for every disorder, were it not due to the disappointing results being reported now, which is putting a check to such early attributions.

As has already been stated elsewhere in this paper Aureomycin is a potent weapon against all rickettsial, many bacterial, certain viral and some parasitic infections. The following conditions have been treated successfully by various workers in the field. For detailed reports of cases you are referred to the Indian and Foreign Medical Journals published recently :

1. Rocky mounted spotted fever.
2. Primary atypical pneumonia.
3. Q. fever.
4. Brucellosis.
5. Typhus.
6. African tick-bite fever.
7. Infectious mononucleosis.
8. Psittacosis.
9. Amœbiasis.
10. Lymphogranuloma venereum.
11. Granuloma inguinale.
12. Peritonitis.
13. Boutonneuse fever.
14. Rickettsial-pox.
15. Pertussis.
16. Sinusitis.
17. Subacute bacterial endocarditis if resistant to Penicillin.
18. Tularemia.
19. Osteomyelitis.
20. Non-specific urethritis.
21. Anthrax.
22. Acute bronchiolitis or pneumonitis of infants.
23. Spirochætes of relapsing fever.
24. Leptospirosis.
25. Gram positive infections including those caused by the following : streptococci, staphylococci, pneumococci, meningococci.
26. Gram negative infections including those caused by the following : coli aerogenes group, and neisseria gonococci.

Aureomycin holds out hope for the following, as preliminary reports are encouraging :

1. Actinomycosis.
2. Acute non-specific pericarditis.
3. Influenzal meningitis.
4. Syphilis.
5. Chancroid.
6. Penicillin-resistant bacteræmia.
7. Bacillary infections of the urinary tract.
8. Herpes Zoster.

Aureomycin has also been found effective in certain ocular infections when used locally and/or orally.

The following are some of the conditions in which it is useful :

1. Conjunctivitis due to various organisms and inclusion bodies.
2. Dendritic ulcers.
3. Superficial punctate keratitis.
4. Epidemic keratoconjunctivitis.
5. Trachoma.
6. Follicular conjunctivitis.
7. Vernal conjunctivitis.
8. Morrens ulcer.
9. Parinaud's conjunctivitis.

Results of treatment with Aureomycin of Salmonella infections are conflicting, some workers have had definitely good response with this therapy while others have not, anyway this confusion is now more or less of academic interest only, for Chloromycetin is more promising.

The above conclusions have been drawn from the work of authorities abroad. Very few reports have been published in India of the usefulness of this antibiotic—perhaps due to its prohibitive cost which has inhibited more extensive trials in the country.

Dosage schedule.—It is as yet too early to assess the exact dosage for an infection susceptible to this drug. However a scheme such as the following (Long[6]) should be a good guide, until more details are available :—

Severity of illness			Initial dose mg./kg of body wt.	Total daily maintenance dose mg/kg of body wt.
Moderate	Oral	...	10	25–50
Severe	Oral	...	20	100
	Intravenous	...	5	15

Note :—(1) The oral priming dose should be split into 3 parts, a part being given at hourly intervals for three doses.

(2) The oral maintenance dose should be split into 6 parts and given 4 hourly.

(3) Intravenous priming dose should be given in one dose.

(4) The intravenous maintenance dose should be split into 3 parts and administered 8 hourly.

The doses for children are slightly less. The therapy may be continued for 5–30 days, depending upon the clinical improvement.

Summary.—Aureomycin has been fully discussed in the light of published reports, with particular reference to its pharmacology. Stress has been laid on the toxic side effects and incompatibility with Aluminium Hydroxide Gel observed by earlier workers, which may have indirectly attributed to its failure, in conditions in which this Gel and Aureomycin were administered simultaneously. The clinical uses of this antibiotic have been detailed and its irrational use has been condemned.

Discussion.—A general discussion followed in which it was observed that this new versatile antibiotic was worthy of trial in amœbic dysentery cases resistant to other forms of therapy, in non-specific urethritis, peritonitis and in typhus fever; in view of the fact that the rickettsial infections in which it is of great use are not very commonly met with in this country.

The dosage schedule was also discussed at length.

The following members took part in the discussion:—Dr. K. N. Udupa, M.S. (MICH.), F.R.C.S. (C.), Civil Surgeon, Mandi; Dr. Jai Lal, B.SC., M.B., B.S.; Dr. K. Pandya, M.B., B.S.; Dr. (Mrs.) Damyanti Kapoor, M.B., B.S.; Dr. H. K. Mathur; and Dr. Chandramani, L.S.M.F.

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Leprosy Relapse after Sulphone Therapy

Paul T. Erickson, Clinical Director of the National Leprosarium, at Carville, reports on six cases of reactivation of leprosy after sulphone therapy and reviews the status of sulphone treated patients whom he followed after apparent arrest of lepromatous leprosy.

Over a 5 year period in which the follow-up varied from 6 months to 5 years after arrest of lepromatous leprosy on sulphone therapy, relapse rates of 45 % for patients not continued on sulphones and 4·5 % for those continued on treatment were experienced. The occurrence of relapse following arrest of the disease, after stoppage of sulphone treatment, indicates that the sulphones are suppressive or bacteriostatic rather than bactericidal in action. The incidence of relapse can be effectively lowered by continuation of sulphone therapy in reduced dosage indefinitely for arrested cases.—(Erickson Paul, T., *P. H. Reports*, 8-9-1950, p. 1147-57).

THIOSEMICARBAZONES—
NEW THERAPEUTIC AGENTS IN TUBERCULOSIS*

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CHEMOTHERAPEUTIC drugs available for the treatment of tuberculosis have no clinical therapeutic effect comparable to the prompt and striking results commonly observed when drugs like Sulphonamide or Penicillin are used in the treatment of other bacterial infections. Of course in human tuberculosis the investigations with possible chemotherapeutic agent present certain difficulties. The *in vitro* activity bears no definite relationship to the *in vivo* activity, and the tuberculosis in laboratory animals again differs considerably from that in man. The recent investigations of Martin (1946), Raleigh and Youmans (1948 *a* and *b*); and Rake *et al* (1949) are, however, offering new approach for the evaluation of tuberculostatic agent on experimental basis. Of the multiplicity of substances recommended for the treatment of human tuberculosis, compounds that have at present won particular importance are the Glucoside Streptomycin, the Para-amino Salicylic Acid and the Thiosemicarbazones.

The antibiotic, Streptomycin, discovered by Waksman, Schtz and Bugic (1944) and first tested by Feldman and Hinshaw (1945) in tuberculosis, and Para-amino Salicylic Acid first synthesized in 1889 (German Patent 50835) and studied by Lehmann (1946) show similar inhibiting effects. But the latter is strongly hindered in its action by p-amino benzoic acid—a growth factor for tubercle bacillus (*Cf.* Ekstrand and Sjogren, 1946); while the Streptomycin is not. The systematic investigations with the Sulpha group of drugs against various pathogenic infections have led to the synthesis of a new group of chemotherapeutic agents that are active against tubercle bacilli. These are the Thiosemicarbazones.

These compounds were also developed in Germany and that again in the laboratories of the Farbenfabriken Bayer at the Elberfeld Works by Behnish, Mietzsch and Schmidt with the co-operation of the discoverer of Sulpha drug, Domagk (1946). Since then a number of compounds in a series have been synthesized and is being evaluated (*Cf.*, Donovick *et al*, 1950; Hamre *et al*, 1950; Francis, Spinks and Stewart, 1950; Basu and Banerjee, 1950). In the series so far the maximal activity in mice is being noticed in p acetylaminobenzaldehyde thiosemicarbazone (I) and p-ethyl sulphonyl benzaldehyde thiosemicarbazone (II).

$$CH_3CO.\ NH.\ C_6H_4.\ CH = N.\ NH.\ CS\ NH_2\ (I)$$

$$C_2H_3 - SO_2\ C_6H_4.\ CH = N.\ NH.\ CS.\ NH_2\ (II)$$

Amongst these two the former has been extensively studied clinically in Germany and very recently in America (*Cf.*, Mertens and Bunge, 1950; Hinshaw and McDermott, 1950; and Tucker, 1950).

* Specially contributed to THE ANTISEPTIC.

Characteristics.—Para-acetaminobenzaldehyde Thiosemicarbazone, more simply known as TB-1, is a pale yellow, finely crystalline powder of bitter taste. It melts with decomposition at about 230°C. It is practically insoluble in water (0·0172 gm. at 37°C). It is more soluble in serum and the solubility is dependent to a considerable amount upon the pH. It is also sparingly soluble in common organic solvents but somewhat soluble in alcoholic caustic soda. The substance dissolves in 50 per cent Sulphuric Acid to about 10 per cent. Antipyrine has the property of solubilizing the substance. The compound can be easily prepared from p-aminobenzaldehyde but its mode of action is not yet clearly established. Its primary action is bacteriostatic. Neither the free Aldehyde group, nor the free Thiosemicarbazide is responsible for its chemotherapeutic action as the free Aldehyde is inactive while the Thiosemicarbazide is very toxic. The acylation, however, plays a special part in imparting the bacteriostatic property to the compound as the free Amino Benzaldehyde Thiosemicarbazone ($NH_2C_6H_4CH = N.NH.CS.NH_2$) is not so active. This is somewhat characteristic in the sense because of the fact that the activity of the product is not inhibited by the presence of Para Amino Benzoic Acid. The drug is rapidly absorbed in the blood system after oral administration and produces some side reactions. As such some of its substitutes by altering its systemic absorption might lower the toxicity of the compound, (*Cf.*, Basu and Banerjee, 1950). The sulfur atom in the molecule seems to be essential as the oxygen analogues (semicarbazone, oxime, hydrazone etc.) of the Aldehyde are inactive (*Cf.*, Behnisch *et al*, 1950). Substitution at the nitrogen atoms of the thiosemicarbazide grouping causes the action to disappear increasingly with the size of the substitutents. It is to be noted that it is the Aldehyde and not ketone and that again of the aromatic nature which afford the active Thiosemicarbazones.

Therapy in humans.—Domagk's observations (1948) on the anti-tuberculous activity of the Thiosemicarbazone (I) *in vitro* and *in vivo* and subsequent findings of Hogarth *et al* (1949) from experiments with animals led to a thorough clinical investigation against various forms of human tuberculosis with this new type of chemotherapeutic agent. In pulmonary tuberculosis the activity of TB–1 shows itself most clearly if the lesion is associated with a perifocal inflammatory process. The Thiosemicarbazone therapy is more promising if the lesions be more labile and if there be better supply of blood. Chronic stabilized cases of pulmonary tuberculosis are found to react less satisfactorily to Thiosemicarbazone medication. Cavernous processes have also shown some response. The findings so far recorded show that the best tendency toward healing is manifested in mucous membrane tuberculosis such as tuberculosis of the bronchus, and laryngeal, intestinal, and bladder tuberculosis. The p-acetylaminobenzaldehyde thiosemicarbazone (TB–1) can also be used as a drug of preference in the treatment of skin tuberculosis but it is contraindicated in miliary and meningeal tuberculosis.

The therapy is not free from toxic effects. At the beginning of the therapy, lack of appetite and gastric complaints are often noticed. But these often disappear with the confirmation of administration of the drug. Skin rashes, conjunctivitis, hæmolytic anæmias and agranulocytosis are other manifestations of toxicity. But if the dosage be properly regulated, most of the above symptoms are seldom noticed and/or easily controlled. There is a tendency toward increased fat deposition in the liver. All these complications should be borne in mind during any therapy with this new agent.

Use of the drug.—The success with this new therapy would depend to a considerable extent on the method of administration, adjustment of the dosage and regularisation of the diet. The dosage must be strictly individualised. In Germany the usual daily dose of TB-1 for adults amounts to approximately 2 mg. per kg. of body weight, by mouth. In any case the dosage must be adapted to the form and the stage of the disease, the responsiveness of the manifestation of tuberculosis, and the individual tolerance of the patient. Further the drug produces a sensitivity toward various substances, particularly to food proteins. A study on the albumin and gamma globulins during therapy might be of some use in controlling the rate of administration of the drug. It is to be noted that TB-1 is not only active for its tuberculostatic activity but also plays some other role either on the blood protein or on the circulating toxin. Most probably this may account for its superior action *in vivo*.

It is being suggested that the therapy should be started with a small dose and slowly increased noting the therapeutic response as well as the individual tolerations. It would be now necessary for our Indian Clinicians to find out the exact dosage for the people of this country. In Western countries the starting dosage is 12·5 mgm. once or twice daily. This is gradually increased to 25 mgm. twice daily.

Researches in this newer direction with this type of compounds might lead to evolution of a better and more tolerate drug.

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SOCIAL ASPECTS OF SKIN DISEASES*

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A DISEASE referable to skin is at times a great handicap in social life. Skin diseases are more common than realized and it is estimated that over 10% of general practice cases are dermatological in nature. Unfortunately, there exist many a misconception about their infection and contagiousness in general public, and these false ideas injure the sufferer psychologically. Patients often have to be taken off work or children excluded from school, not because there is any danger in continuing there but because of feelings engendered in others. Leucoderma (white spots in the skin) is quite common and it is unluckily often regarded as "leprosy", and the poor patient is shunned and neglected. When such is the fate of one suffering from this innocent condition, the laity's reaction toward actual leprosy, which is quite rife in our country, could not be grasped. A man suffering from leprosy is called by such name as "Leper" and he is looked down and thrown to the streets.

Just to quote another familiar condition is "acne vulgaris", the so-called pimples, and these make the sufferer to keep off from social engagements. This condition is always taken as "something to do with the youth" or "something bad with the blood". Hence the shelter of "blood purifiers" is taken and they usually contain such drugs as Bromides or Iodides which make the condition still worse and give rise to "drug rash". Boils (furunculosis) and prickly heat are also regarded as "due to bad blood" or "heat in blood" and for these a person sometimes entreats for purgative to clean his body internally rather than using local medication.

One can go on summing up many skin conditions regarding which many false ideas are there in public. Just in passing, another instance is *Herpes Zoster* (or Shingles), which is regarded as "following the visit of a spider on the skin."

As for other examples, one may mention "Small-pox" about which such erroneous ideas are prevalent in lay public that it is regarded as a divine curse or displeasure and the patient is nursed in the most filthy circumstances and thus permanent and unwelcome damage is done to the skin. Any generalised skin rash is often mistaken for "*Mata*" (*i.e.*, small-pox) which, on careful scrutiny, may be just manifestation of syphilis. Conversely, many generalised skin conditions are often regarded as secondary to syphilis when injudicious treatment employing active arsenical drug causes extensive damage to the skin along with damage to liver, kidney, etc. The idea that many a drug when ingested by mouth may bring about generalised skin rash is hardly conceived.

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* Specially contributed to THE ANTISEPTIC.

It is truly said that a sound health is essential for a sound mind. To maintain a perfect health, skin cleanliness is the first thing. If one suffers from scabies or lousiness which are associated with overcrowding, lack of facilities for personal hygiene and cleanliness, his efficiency for work diminishes, as he cannot have a restful repose. The incidence of these diseases goes up under conditions of war and stresses during the refugee influx over here in 1947-'48. The number of loss of "man-hour-work" brought about by these is incalculable. Hence, there should be available facilities for treatment etc., for these conditions at public places at the cost of the Government. In many countries *viz.*, England and Denmark, such treatment centres exist and thus they have controlled these infections. Lousiness should be effectively treated as this is responsible for typhus fever.

Many skin diseases are spread from person to person and this goes on unknowingly. Dandruff is quite a common condition and it spreads through combs and brushes which are usually in common use in a family. The barbers and hair-dressers who keep their combs and other tools in an unclean condition, play an important role in the spread of this condition. Dandruff may persist throughout the life time without causing any symptom, but at times it may cause widespread dermatitis. If it be neglected, it may give rise to baldness.

To improve the health of our nation, our people should have their attention drawn to health problems. The public should be well informed and so health education is all the more important. Similarly, they should know how to protect their skin which is an equally important organ of the body as it is the first line of defence, and, through it, one keeps alive to the external world. Any skin condition should not be lightly regarded but proper care should be taken. Skin disease is visible and treatment seemingly successful. Thus the patient and doctor can both observe progress directly and derive pleasure from good results and both are satisfied. But in spite of this, self treatment and self cure are quite rife.

It is impossible to imagine how many skin diseases are self-treated. Itching or irritation is a common feature of a good number of skin diseases which usually have different causation. To add to our misfortune, there is no dearth of advertised medicines which contain various strong chemicals in varying proportions and these act as sensitizers and give rise to "dermatitis medicamentosa" *i.e.* harm produced to the skin by the medicine, and this unfortunately is more troublesome and agonizing as compared to the original skin condition. If you just pass along a market, you will find advertisements stating that such an ointment is good for all skin conditions *viz.*, eczema, scabies, ringworm etc., etc. Peculiar ways of display of the advertisement are there to catch the public eye. It is necessary that the Government should take proper steps to stop such

malpractice which brings untold miseries to many. The "Quacks" should be banned.

Skin and central nervous system are closely related developmentally as being originated from the same structure (epiblast). So skin is very much influenced by environment *e.g.*, economic instability and other adverse social factors. As for instance, infantile eczema, being intractable, presents a problem to the mother who sees her child suffering from intense paroxysms of irritation which often makes the baby to scratch until the skin bleeds. As weeks and months go by with little improvement, the mother may show signs of breaking down.

Cosmetics (face or hand creams, lipstick, face powder, nail polish, etc., etc.) are a boon to skin as they go a long way to save a person from a social trauma and makes him or her befitting for a society. These widely used in times of antiquity are now an integral part of the lives of civilized communities and of all classes. They enhance or add to personal appearances and correct minor blemishes and also reduce the effect of work, Sun, wind and sweat upon the skin. Lipsticks prevent cracking and chapping of lips caused by wind. But in some sensitive persons, they may give rise to various chemical dermatitis.

As we hope to progress in industries, there should be an industrial medical service enabling industry to provide medical care of workman at his job and to concentrate on control of industrial and occupational diseases on the spot. It would pay the various industries to seek the advice of a dermatologist before employing a person in a particular trade. Besides this, there should be posters displayed in prominent places in the premises of the factory, where various chemicals are used to save the employees from getting chemical or industrial dermatitis. A medical officer employed in a factory should have a thorough knowledge of various industrial diseases. Thus the various industrial diseases pertaining to the skin could be diagnosed in the early treatable stage.

The fact that some skin diseases of domestic pets *e.g.*, cats, dogs, horse and cattle may also be transmitted to human beings, should also be recognised. Ringworm thus acquired may affect nails of the fingers of hand besides affecting the smooth skin and thus render one shy to shake hands.

The idea that skin troubles are only cured by ointments should be purged out of the public mind. Undoubtedly, ointments are mostly used. But there are many ways of applying medicine to skin *viz.*, paints, lotions, powders etc., etc. Electrotherapy plays an important role in the treatment of skin diseases. Remember that many skin diseases may be just a manifestation of some internal cause, *e.g.*, deficiency or irregularity of diet. An incipient diabetes may give rise to chronic itch.

Attempts should be made to attract the public to hospitals for the treatment of skin conditions for which there should be no feeling

of any social stigma as skin and V. D. are usually tackled together in some clinics. The patients feel diffident to attend them as they might be singled out to be suffering from V. D.

Skin may be regarded just a mirror upon which effects of dirty environment, poor, unbalanced diet, inefficient evacuation and septic focal spots are exhibited.

VITAMINS*

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THE knowledge about vitamins has made enormous progress since the days of Sir James Lanchaster in 1601 when he first introduced fresh oranges in the diet of his naval ratings to prevent a certain disability they suffered from, when on long voyages, which we today know as scurvy. It was all trial and error. Yet that opened out a wide lacuna of our knowledge of vitamins. The progress for further research had to be slow. No wonder therefore that the next in order of these essential factors was not discovered till about 1885. It was Takaki this time, working with the Japanese Navy, who noticed that his men when fed on polished rice showed symptoms of a disease which we today know as beri beri. This gave impetus to fresh work and diseases whose origin was obscure were viewed in this new light. So it was that towards the end of the last century that some pediatricians suspected that rickets had some such origin—deficiency of an essential substance. But it was not till the beginning of this century that real experimental work began on this research. It was Eijkman in the East Indies who produced experimental beri beri in fowls. Girgins, his collaborator, isolated the antineuritic substance from rice polishings. Yet, that these essential substances could be vivisected and synthesized was never dreamt of as possible previously. They knew that these essential substances existed in certain foodstuffs. Funk worked over these substances for a long time and first propounded his vitamin theory. He postulated the existence of separate essential substances in certain commodities of food, absence or destruction of which produced a certain specific disease. Eijkman and Hopkins working separately proved this hypothetical theory by animal and later human experiments. They were therefore awarded the Nobel Prize in 1929.

2. Initially these vitamins had been very few and simple. Deficiency of any of these gave rise to a clearly defined disease. Today as many of these vitamins are known as there are letters in the Roman Alphabet. Many of them are known to form a complex and their deficiency gives rise to intricate syndromes. In the last few years biochemistry trying to be helpful essayed simplification

* Specially contributed to THE ANTISEPTIC.

by fractionation of certain of these complexes. Today we have this in vitamin 'B' complex, a collection of major and minor vitamins. A general practitioner has a hard time finding his way out of these. A brief resume of all these vitamins with their uses and limitations is therefore here attempted.

3. All these essential substances or accessory food factors or otherwise known as vitamins, are broadly classified into two : (1) Those that are present in fats. They are fat soluble. Fat soluble vitamins are vitamins A, D, E and K. They are found in most animal and vegetable fats. (2) The water soluble vitamins comprise the vitamin 'B' group and the vitamin 'C'. I shall not bother you with certain other vitamins whose existence and usefulness in human beings is still in dispute.

4. Broadly speaking all these vitamins have three main physiological functions :—

(*a*) Most of them are essential for normal growth development and maintenance of health.

(*b*) Their deficiency produces specific diseases which easily are curable when that specific vitamin is administered.

(*c*) They are used as adjuvants in the treatment of conditions which are not primarily deficiency diseases but which are likely to result in them.

5. Apart from this, there has been taking place an insidious development in vitaminology which may eventually lead to a division of this branch of therapeutics into two sections.

(*a*) Vitamin enzymotherapy where vitamins are used for the prevention and treatment of deficiency conditions in a relatively small amount. Most of the water soluble vitamins come under this category, with the exception of vitamin 'B_1' and 'C' which belong to this as well as to the next branch.

(*b*) Vitamin pharmacodynamic therapy where massive doses are given in order to utilize their potent metabolic effects in the prevention and treatment of diseases. The fat soluble vitamins fall under this branch. More will be said about these actions while dealing with individual vitamins.

Vitamin 'A'.—Also known as anti-ophthalmic and resistance builder. It is a fat soluble vitamin and is present in most edible oils, fats of animals and milk. It is found in a very concentrated form in the livers of cod and shark fishes. A provitamin of these vitamins is found in most leafy vegetables. As a matter of fact the more green a vegetable is, the more rich are its contents of B. c arotene (Provitamin 'A'). The lower animals mostly synthesize these vitamins in their bodies from the grass they eat. The man, however, has to be supplied with this ready product in his food. Liver is the chief storehouse of this vitamin and that is why gross deficiency of this vitamin is rarely seen.

6. Deficiency of this vitamin leads to the following conditions :–(*a*) Night blindness; (*b*) failure of growth; (*c*) xerosis of epithelial tissues or metaplasia; (*d*) multiple secondary infection; (*e*) xerophthalmia; (*f*) keratomalacia; (*g*) renal calculi; (*h*) peridental hyperplasia; (*i*) reproduction failure; and (*j*) degeneration changes in central and peripheral nervous system.

In one individual case one may not notice all these conditions. It depends on age and at what stage of deficiency the case is seen. In a few deficiency cases one sees most of the above conditions. The most sensitive test for detecting hypovitaminosis of this vitamin, is by dark adaptation. It is wise to make use of this vitamin as a prophylactic measure in small infants and growing children and in those who are confined to bed, or those who are on restricted diets. The prophylactic dose is 5,000 to 10,000 I.U. daily. In certain skin diseases (acne vulgaris, seborrheic dermatitis, keratosis follicularis), mucous membrane disorders, for example respiratory and gastro-intestinal disturbances; rhinology *e.g.* for treatment of *ozena*, in ophthalmology (xerophthalmia and keratomalacia), massive doses have to be given. These doses range between 1,00,000 I.U. to 6,00,000 I.U. daily either orally or parenterally. For those who are intolerant to fats, water soluble vitamin 'A' is also available and this must be used in preference to the other where the digestive system is not normal.

Vitamin 'D'—Also known as anti-rickets vitamin. This is present in most animal fats. The richest sources are cod and shark livers. Recently methods have been prefected for preparing this vitamin synthetically. It is known as Calciferol which is prepared by the ultraviolet irradiation of Ergosterol. There are many Sterols prevalent in nature and with suitable irradiation they can be converted into any of the vitamins 'D'. At least three of these are known. The action of all these is the same, control of calcium metabolism. Vitamin 'D' causes increased net absorption of calcium from the intestinal canal. Its deficiency causes disturbed absorption and metabolism of calcium and phosphorus resulting in rickets in young life and osteomalacia in the adult. Infantile tetany is a nutritional disorder and its direct cause is hypocalcæmia. It is not due to malfunction of parathyroid as some believe it to be, and its (parathyroid) administration in the initial stages aggravates the condition by mobilising bone calcium. Failure to absorb fat sometimes causes rickets and administration of vitamin 'D' in a non fatty vehicle cures the condition. Optimal prophylactic dose of vitamin 'D' for infants and children is 500 to 1000 I. U. Curative dose is 1000 to 3000 I.U. and the optimum dose is 10,000 I. U. Unlike other vitamins this vitamin cannot be given *ad lib.* The maximum 10,000 I.U. should not therefore be exceeded.

Calciferol (Vitamin 'D') marks a spectacular advance in therapy of lupus vulgaris. It has to be given in massive doses, remaining clear always of the optimal dose.

Vitamin 'E'.—Also known as anti-sterility factor or wheat-germ oil occurs in the oil from wheat-germ, rice-germ, cotton seed, maize and in green leaves. It has been isolated from these natural sources as Tocopherol. The Alphatocopherol is the richest, the *Beta* and *Gamma* being poorer in their activity.

Lack of vitamin 'E' produces different lesions in the two sexes; in the male it produces gradual atrophy of testis with degeneration of the germinal epithelium, and in the female it leads to characteristic type of secondary sterility where a normal pregnancy is abruptly terminated either by the death or absorption of the fœtus. Like vitamin 'A' Tocopherol is also stored in the liver and therefore deficiencies of this vitamin very rarely occur. Prophylactically it can always be given in 10 to 30 mg. doses. In habitual abortion where all organic causes have been excluded massive doses of this vitamin (100 mg.) are necessary. For sterility in the males due to faulty spermatogenesis the prophylactic doses 10 to 30 mg. will be sufficient. Vitamin 'E' is considered to have pharmacodynamic action on conditions sharing common sclerovascular pathology *e.g.*, intermittant claudication, Dupuyetrin's contracture, fibrositis, angina pectoris etc. The usual dose is 300 to 600 mg. daily.

Recent claims that neuromuscular diseases such as muscular dystrophies, progressive atrophy, amyotrophic lateral sclerosis, and aftereffects of poliomyelitis are cured or improved by this vitamin have not been substantiated.

Vitamin 'K' is a fat soluble vitamin and promotes formation of prothrombin in the liver. Prothrombin is essential to the normal coagulation of the blood.

It is absorbed from the intestinal tract in the presence of bile salts only. Conditions like obstructive jaundice, diarrhœal conditions after operation requiring lengthy resections of intestine, and habitual medication with liquid paraffin, produce hypovitaminosis 'K'. Since liver plays an important part in its synthesis, it is obvious in conditions where liver tissue is damaged signs of hypovitaminosis 'K' should make their appearance. Its deficiency can be measured by the degree of hypoprothrombinæmia by Quick's test (measuring coagulation time).

Little is known about its total requirements. For therapeutic dosage, the prothrombin level is a good indicator. Infants require about 2 mg. s.o.s. and adults 10 and 20 mg. s.o.s.

It is of use in all hæmorrhagic disorders (not in hæmophylic purpura), in disturbances of fat absorption (sprue, cœliac diseases etc.) in liver diseases such as cirrhosis of the liver, hepatitis, obstructive jaundice, and in hæmorrhagic diseases of the new born *e.g.*, umbilical and subconjunctival hæmorrhages etc.

Water soluble vitamins, as already said, consist of the vitamin 'B' complex group and Vitamin 'C'. They are not stored to any extent

in the body. They therefore have to be supplemented or eaten with food. Their deficiencies are more frequent and varied.

Vitamin 'B' complex group comprises of thermolabile and thermostabile components. The thermolabile are further essayed into Vitamin B_1 and Pantothenic Acid. Both of these do not withstand heating.

Vitamin B_1.—Anti-beri beri factor - Aneurin or Thiamin Hydrochloride. This was isolated many years ago from rice polishings. It is also prepared synthetically and is known as Thiamin Hydrochloride. It occurs in high concentration in the outside bran coats of cereals. It is abundantly present in Brewer's yeast.

Its deficiency produces beri beri, the principal features of which are anorexia, marasmus, cardiac abnormalities, paresis or paralysis and sometimes œdema. Its characteristic nerve lesion is a demyelination of peritheral nerve, the so called polyneuritis of the alcoholic and pregnancy. It has an important physiological action–the enzyme vitamin function referred to already. It provides a co-enzyme necessary in the breakdown of pyruvic acid in the intermediate metabolism of carbohydrate to carboxylase.

The daily requirements of this vitamin depends upon the carbohydrate in-take. Generally about 1–2 mg. are sufficient. Massive doses are, however, required while treating polyneuritis and other frank deficiency conditions. In combination with Nicotinic Acid Vitamin B_1 is used successfully in delirium tremens in alcoholics. (About 100 to 300 mg. of Thiamin with 50 to 60 mg. of Nicotinic Acid repeated s. o. s.)

Pantothenic Acid.—The filtrate factor or the anti-grey factor (also known as chick anti-dermatitis factor) is still in the experimental stage of discovery. It is supposed to be essential for the development and natural functioning of the tissues. Its deficiency leads to disturbed metabolism and diminished resistance of epithelial and mucosal cells (especially of the respiratory and digestive tracks).

Its daily requirement in human beings is not known. For deficiency signs about 50 to 100 mg. of the salt are daily required. Owing to its epithelial cell-regenerating property it is also used as a lotion in burns or surgical wounds.

The thermostabile component of the vitamin 'B' group consists of: (1) Nicotinic Acid; (2) Lactoflavin or Riboflavin; (3) Pyridoxin; (4) Biotin; (5) Para-amino Benzoic Acid; (6) Insitol; (7) Folic Acid; (8) B. 12; and (9) Choline.

Nicotinic Acid, also known as anti-pellagra factor or P. P. factor, is known since many years. It plays an essential part in the body enzyme reaction.

Its frank deficiency produces pellagra—known by the three D's: Diarrhœa, Dementia and Dermatitis, though in all cases the

three may not be necessarily present. The cutaneous lesions produced by its deficiency are symmetric pigmentation of the skin which may progress to atrophy of the skin as deficiency increases. Nervousness, tendency to undue fatigue, insomnia are some of the early symptoms of its deficiency.

The average daily requirement is estimated to be 10 to 30 mg. For treating deficiency conditions large doses upto 400 mg. per day may have to be used. It is said to minimise the toxic effects of Sulphonamides. As already said, it is used in conjunction with vitamin B_1 in the treatment of delirium tremens (alcoholic).

Lactoflavin also known as Riboflavin is the yellow constituent of the B complex group. It is so widely spread in the animal and vegetable kingdom and being relatively heatstabile, a frank deficiency of this vitamin should not occur. In those cases where it is seen it is usually due to incomplete absorption or intermediate destruction. In pregnancy there is an increased demand of this vitamin and deficiency symptoms might occur in that condition.

This vitamin is essential to a number of vital oxidation-reduction processes concerned with the metabolism of carbohydrates, proteins and fats. It enters into the structure of a large number of enzymes taking part in tissue respirations. It helps dehydrogenation of citric, malic and lactic acids in the muscles, oxidises aldehydes as in liver and milk, and deamines histamine. Thus probably it plays some part in the prevention of certain allergic states.

Its deficiency produces oral, skin and eye lesions. The cheilosis, angular conjunctivitis, sometimes with circumcorneal injection, and desquamation with crust formation, as in seborrhœic dermatitis, are very characteristic lesions of this deficiency. The daily estimated dose is about 1·5 mg. The usual dose in any deficiency is about 10 to 20 mg. There is no fear of hypervitaminosis with Riboflavin, as any excess of this vitamin is excreted giving the flavin colour to the urine.

Pyridoxin.—Also known as Adermin or Vitamin B_6. Its physiological role in man is not clearly understood. It is reported to have a marked sedative effect in man. Its deficiency probably leads to hyper irritability.

The human daily requirement is unknown. It is widely distributed in chemical combination with foods and 1 mg. is suggested as the daily dose for human beings.

Biotin.—Some time back called as Vitamin H. This is the latest of these series to be synthesized. It is a growth factor for bacteria (intestinal).

Its deficiency can be experimentally produced by feeding animals on white of eggs. It also results while on treatment with Sulphaguanidine or Sulphathiazole in certain persons. Its lesions

are a generalised scaly dermatitis, worst on the face, arms and legs. Its daily requirements are not known. Its experimental dose in human beings is about 150 to 300 microgrammes daily.

Para-amino-Benzoic Acid.—It is also a growth factor for many bacteria in the intestines. It is by depriving this food to bacteria that Sulpha drugs exert their bacterio-static action. Para-amino-Benzoic Acid is one of the constituents (bacterial) in fresh buttermilk and this explains why sometimes one finds Sulpha drugs quite inactive when a person is consuming large quantities of buttermilk or curds. The beneficial action of buttermilk in certain diarrhoeas may also be due to its being a vehicle for introducing the right flora and their food into the human intestines. For man therefore this vitamin component is only a secondary nutrient.

Its daily requirement is not known as also its dosage. Some experimental work is being carried out in this country for its use on human rickettsial diseases, particularly typhus.

Insitol.—So far its action is only known in lower animals. Its deficiency in them causes hairlessness and severe dermatitis. It is so widely distributed in plant and animal life that its deficiency probably never occurs. Like Para-amino-Benzoic Acid it also acts as a second nutrient, since its presence is essential for the biological activities for certain intestinal flora.

Minimum requirements in human beings are not known and even very large doses do not seem to do any harm.

Folic Acid.—Known also as vitamin M, L. Casei factor or Pteroylglutamic Acid. It is called Folic Acid, because it is found in green foliage. It is also present in yeast, animal tissues, liver and kidney. This is normally synthesised by bacteria present in the human intestine. The growth and development of this bacteria forming the flora, however, depends upon the available Folic Acid in a person's diet. It would be reasonable to expect, therefore, that in conditions where this is in short supply or in conditions, where an abnormally large number of bacteria are present in the upper part of the small intestine, a deficiency of this vitamin occurs. The latter is supposed to be a more common condition and possibly a cause of the sprue-syndrome. In this disease an invasion of bacteria is supposed to occur in large numbers and this accounts for the large bulky fermentating stools and the extra amount of Folic Acid required to sustain these additional invaders. This fits in with our clinical experience that this complex improves after parenteral administration of Folic Acid, or where large doses of the same drug are given by mouth. The metabolic demands of varying but essentially nonpathogenic flora may be the key to the problem of the multiple but selective vitamin deficiencies so commonly observed in these conditions.

Its deficiency produces the sprue syndrome. It is besides very useful in such purely nutritional aspects of deficiencies as gastro-enteritis, glossitis, stomatitis, steatorrhœa and hypoproteinæmia. Its chief interest is however in megaloblastic anæmia and leucopenia. Its unrestricted use sometimes precipitates neurological complications of an irreversible type. Its use therefore in Addisonian type of anæmias should be carefully watched. Its daily requirement in man are still uncertain. For therapeutic action about 5 to 30 mg. are sometimes required. The exact dose is an individual factor. The maintenance dose is usually very small. The synthetic preparations of Folic Acid are supposed to be even superior to the conjugated form of Folic Acid which is interesting, because much of the Folic Acid in nature, therefore, in the human diet, is in this state.

Vitamin B_{12}.—This is a very recent number of the B complex vitamins. As is usual with all recent discoveries a hell of a noise is made about it, some even call it the last remedy for all types of anæmias. Time however is the best test for all such remedies and a wise clinician always depends on this for his choice.

This was originally isolated from crude liver extract by Leslie Smith. It promotes the growth of Lacto bacillus lactis Dorner—the L.L.D. factor. It induces hæmopoietic response in patients suffering from pernicious anæmia. The reticulocyte response is very brisk and reaches its peak in about 4 to 9 days. Unlike Folic Acid this is remarkable in its effect in relieving subacute combined degeneration in pernicious anæmia patients. It is not so effective by mouth in the initial stages of the disease since it is supposed that the intrinsic factor in the gastric juice is necessary for its absorption. Large amounts of this vitamin have been isolated from human fæces in untreated cases. It is therefore given by injection, or if the oral method is to be preferred very large doses have to be given (450 microgrammes). In uncomplicated cases 10 to 15 microgrammes are required once or twice a week (roughly one microgramme a day). An effective maintenance dose is again an individual factor. For arresting subacute combined degeneration of spinal cord about 30 microgrammes once or twice a week are required. It is useful in quite small doses (a single dose of 10 microgrammes) in nutritional macrocytic anæmia, in sprue and in macrocytic anæmia of infancy (a single dose of 25 microgrammes).

Choline or Choline Chloride.—It is still under clinical trial. It is supposed to play a useful part in the transport of fat from the liver and therefore theoretically would be an ideal remedy for fatty infiltration of the liver. It cannot be given *ad lib.* The toxic dose lies very near the therapeutic dose which is about 1·5 grams. which should not be exceeded.

Vitamin 'C'.—Anti-scorbutic vitamin or Ascorbic Acid. This is the first vitamin whose presence stimulated the research of similar food adjuncts. It is thermolabile but the vitamin 'C' present in

citrous fruits is peculiarly stable. In "amla" or the Indian gooseberry, it is retained even when the fruit is dried. For a strong physiological functioning of all the systems of the body, adequate quantities of this vitamin are essential. It acts as an oxido-reductive substance regulating cellular respiration. It is essential for the maturation and growth of red cells, white cells and platelets. It has some action in stimulating the healing of wounds, probably by helping to build up interstitial tissue. It prevents capillary fragility and in consequence protects one from capillary hæmorrhages (Petechiæ). It is an anti-infective factor. It is also anti-toxic and desensitizing agent. It has been alleged that this vitamin is also concerned in the production of anti-bodies.

Its deficiency produces the typical scurvy—easily bleeding gums, tendency to petechiæ, and capillary bleeding etc.

The early signs of hypovitaminosis 'C' are undue fatigue and a feeling of lassitude, tendency to cough and colds, decayed teeth and spongy gums, slow healing of wounds or fractures. Certain conditions such as pregnancy, lactation, periods of change in life, and acute infections, produce an extra demand for this vitamin. The body stores a certain reserve of this vitamin but an excess is easily excreted in the urine. This fact is utilised for measuring 'C' hypovitaminosis and the severity of an infection (which is inversely proportional to the infection). The suprarenal cortex probably acts as a store house for this vitamin.

The daily requirement of this vitamin differs at different ages, in health and disease, and at rest or while exercising. An infant requires about 25 to 30 mg., a growing child 75 mg. upto 12 years, and an adult requires 75 to 85 mg. Twice these amounts are required in febrile conditions in the different age groups and about 100 to 150 mg. in pregnancy and lactation.

Conclusion.—Vitamins have a great future. Their bio-synthesis is now known very well. They are not merely accessory food factors. They play an essentially vital role. They are necessary for tissue respiration, for certain oxidation reduction processes, and also in their enzymic action. Their competitive phenomenon is now better understood. Nobody therefore prescribes large supplements of single vitamin. The pendulum has once again swung back and we have again begun prescribing the old blunderbuss form of treatment.

They have opened still another apartment for research work. Some workers have found an excess of some of these vitamins in certain malignant new growths. Whether this is just an accidental finding or whether depletion of that particular vitamin would stop or cure the condition the future alone should tell.

There is also another interesting finding connected with vitamins. Some of them *e.g.*, Ergosterol (Vitamin D_2) has a sterol

nucleus. So also have the human sexual hormones—Testoviron—Stilbœstrol. I have already told you about the supposed presence in some good amount of vitamin 'C' in the adrenal cortex. Does this show any correlation ? Are the vitamins just extrinsic hormones? These are difficult questions to answer. We can think in the present. We only dream about tomorrow.

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DIVES INTO ANCIENT HINDU MEDICINE

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(Continued from page 379 of May '50 issue).

Section IV. Kapha-Masurikas

THESE too, like Vaata- and Piththa-Masurikas hold six main types; each main type holding sub-types, there are 32 kinds of Kapha-Masurikas. General and constitutional features have been discussed in Section I; the individual characteristics of the different kinds will be considered here.

MAIN VARIETY	SUB-VARIETIES
I. Paaloori (5 minors)	(1) Paaloori proper, (2) Manal-paaloori, (3) Ari-ppaaloori, (4) Viyarp-paaloori, (5) Ninal-paaloori
II. Vin-chilanni (6 minors)	(1) Venchilanni proper, (2) Vel-chilanni, (3) Aana-chilanni, (4) Vatta-chilanni, (5) Vitta-chilanni, (6) Kootta-chilanni
III. Moar-chilanni (5 minors)	(1) Moar-chilanni, (2) Paal-chilanni, (3) Kaal-chilanni (4) Thati-chilanni, (5) Vetti-chilanni,
IV. Neer-chilanni (6 minors)	(1) Neer-chilanni, (2) Koor-chilanni, (3) Pon-chilanni, (4) Aar-chilanni, (5) Poar-chilanni, (6) Veer-chilanni.
V. Pon-chilanni (5 minors)	(1) Pon-chilanni, (2) Mani-chilanni, (3) Maanikka-chilanni, (4) Aani-chilanni, (5) Kata-chilanni.
VI. Oatu-chilanni (5 minors)	(1) Oatu-chilanni, (2) Ul-chilanni, (3) Kaattu-chilanni, (4) Kaal-chilanni, (5) Maattu-chilanni.

I. **Paaloori.**—The affection is so called probably because of the odour of cow's milk given out by the efflorescences when ripe. There are 5 sub-groups in the affection; only one of them proves fatal. The presiding devatha is "Pulli-Marutha."

CLINICAL FEATURES:—1. General characteristics are as in Kapha-jwara in Masurika, (Section I). Fainting or unconsciousness at onset of fever, intense thudding head-ache, congested eyes; vomiting constantly, and diarrhœa rarely; general pains over and

a swollen condition of body—these are some of the main features observed in all varieties of Paaloori.

All types of Paaloori, especially the Paaloori proper, are highly infectious; it is said that an infected case quadruples the number in the affected group by infecting three more.

2. Special diagnostic features: (*a*) If, during the height of fever, the nasal discharge of the patient is thrown into the juice of green turmeric, the whole will turn blood-red in about half an hour (one 'naazhika').* (*b*) Mixture of patient's urine and breast-milk, if kept undisturbed and examined after one naazhika, will exhibit bead-like formations.† (*c*) During the prodromal period, application to umbilical region of a paste of leaf-buds of Ficus Religiosa will make the rashes visible—although the eruptions normally come out only a day or two afterwards.

CLINICAL MANIFESTATIONS:—1. *Paaloori proper*: General features have been discussed above. The rashes, when they appear on the third day, are pepper-sized, red-topped, and hemispherical or triangular. They become white on the 7th day (of fever or eruption ?); gradually ripen, emitting the odour of cow's milk; and break leaving white scabs.

The patient can be bathed after 14½ days.

2. *Manal-paaloori*:—('Manal'=sand):—Resembles the above, except in the rashes being smaller. Patient can be bathed on the 10th day; if the patient be strong, bath may have to be delayed to the 14th day.

[It was mentioned under Poozhi-kkalloori (Section III) that there permeates about the patient the sweet odour of Champak flower when the rashes break, provided the patient is *not* strong ('Sakthan'), in which case there will be a mal-odour surrounding him. And in Manal-paaloori we are told that the stronger the patient the more delayed will be the day of bath. Can the conclusion be "the greater the vitality, the more the pabulum offered to the invader; and consequently, the greater the vitality the more offensive the pathological products of 'war', or the greater the inducement for the invader to stay and despoil the host" ?]

3. *Ari-ppaaloori*:—('Ari'=rice):—The rashes here are bright and clear and of the size and shape of rice grain; and they continue to be bright and clear when they ripen too. When the efflorescences get resolved, the eyes become congested—a special feature of this affection.

The patient can be bathed on or after the 14th day.

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* Juice of green turmeric—for that, a solution of dry turmeric too—will turn blood-red on the addition to it of a small pellet of 'chunnam' or slaked lime.

† Apparently, the bead-like formations are curds or clots.

4. *Viyarp-paaloori* :—'Viyarpu'=sweat:—The 'Birth' of the rashes outside is accompanied by profuse sweats all over the body. The patient can be bathed on or after the 12th day.

This affection is also called 'Visarp-paaloori.'

5. *Ninal-ppaaloori* :—The rashes, when they appear, are sand-like in size and shape and blood-red in colour. Nasal discharge will be in plenty ; and, at some time or other, there will appear blood in urine and motions. On the 6th day (of fever ?), the rashes recede ; between the 6th and the 9th day, the eyes are affected and become blind ; and on the day the eyes get involved, the patient dies. Death is usually sudden and occurs just after 8½ days from the day of onset.

TREATMENT.—1. *Externally* :—As indicated.

2. *Internally* :—(*a*) *Cooling oushadhas* : (i) the spiritous liquor called 'Sura'. (*b*) *Decoctions* : (i) onions, coriander, Bael root, Laaja powder—prepare decoction, and sign as usual; and (ii) decoction of Naal-paal-mara.

3. *For the eyes* :—Cumin seeds, flower buds of Tulsi, kernel of very tender cocoanut—bundle, crush, soak in breast-milk and squeeze drops into eyes.

II. **Ven-Chilanni.**—'Chilanni', also known as 'Chilanthi', means 'spider'. Perhaps, the particular affections of variola with their appellations ending in Chilann (th) i were called so because of the eruptions resembling a spider in appearance at one stage or the other of the disease.

Ven-Chilanni holds 6 sub-groups, of which all except "Vatta-chilanni" are of very grave prognosis ; Ven-chilanni proper, Vittu-chilanni, and Kootta-chilanni invariably prove fatal, while cases of Vel-chilanni and Aana-chilanni have a remote chance of recovery. Cases associated with sweet odour of lotus in the early stages and mal-odour later are of comparatively good prognosis ; cases with following features rarely recover—prolonged unconsciousness at onset, breaking of rashes delayed to after 14½ days and bleeding into the raw surfaces then, anuria or retention of urine ('Moothra-sth-thambhanam"), involvement of eyes, laryngeal or pharyngeal obstruction, raving mania, and terminal diarrhœa. Cases that recover do so with loss of function of one or more of "Chathur-Indriyas," *viz.* eyes, ears, nose or tongue, or some other sequela.

The presiding devatha is 'Kolli—Marutha'—a fiery Marutha indeed ! ('Kolli'=fire or incendiary).

CLINICAL FEATURES.—1. General features as in Kapha-Jwara of Masurika (Section I).

2. Special diagnostic features : (*a*) During the acme of fever, the patient's hair, if thrown into water, will gradually sink to the bottom ; (*b*) application of a paste of Gorochan in tender cocoanut

water to the soles will make rashes on the forehead visible, during the prodromal period.

CLINICAL MANIFESTATIONS.—1. *Ven-chilanni proper* :—General features are as in Kapha-Masurikas and are seen in all varieties of Ven-chilanni—Loss of consciousness at onset, intense headache, congested eyes, vomiting, severe constipation, nasal discharge, sweats on face—these are the main symptoms and signs.

The rashes, when they appear on the third day, are pretty large and lotus-bud-like with a broad circular base and a pointed apex. The apices, as the rashes grow, get less and less pointed and more and more flat till the eruptions in course of time break and dehisce even as lotus buds do into lotus-flower; and then, there will be given out an intense foul odour. The patient invariably dies on the 18th day.

2. *Vel-chilanni* :—The rashes, when they appear, resemble those of Ven-chilanni, but show red streaks all round their bases (sign of inflammation ?). They do not dehisce, but get fissured; the open wounds are whitish or bloody. In the former event, the patient can be bathed after 22½ days; in the latter case, the patient dies within or close after 22½ days.

3. *Aana-chilanni* :—'Aana'=elephant. The eruptions here, as the name will indicate, are very large. Except in size, they resemble Ven-chilanni rashes. When they break and begin to desiccate, they leave large black open wounds which, appearing over the whole body, give the patient a hideous appearance.

The prognosis is bad; in exceptional cases, the patient may recover and be bathed after 38½ days. But, it will take 90 days for the body to be clear of all skin-wounds.

4. *Vatta-chilanni* :—The eruptions here are circular. When they break, they do so discharging much blood and pus. The patient usually dies within 18 days; if death does not supervene within the 18 days, he can be bathed after 18½ days.

5. *Vittu-chilanni* :—When the rashes come out, there appears diarrhœa too. And when they break, there sets in mania and the patient develops pica—a morbid appetite for the most revolting inedible materials, including excreta. The patient invariably dies on the 25th day.

6. *Kootta-chilanni* :—The rashes here are peculiar in that they come out in clusters of bubbles. Another peculiarity about them is that they so configure themselves that they look like human hands and feet to the observer. When they break, they profusely bleed. The patient dies on the 20th day.

TREATMENT.—1. *Externally* :—As indicated.

2. *Internally* :—(*a*) Solution through boiling of pistils of lotus flower in tender cocoanut water; (*b*) decoction of liquorice; (*c*) red sandal, China rose, onions and "Makki-poo"—in equal

parts, prepare decoction and sign as usual; (*d*) decoction of Chob Chini purified through boiling in milk; (*e*) kernel of seeds of Mimusops Elengi (Bakula), Saussurea lappa, Acorus Calamus, Nigella Sativa—in equal parts, powder finely and give with honey as linctus frequently or in measured doses at fixed intervals; and (*f*) give with honey fine powder of Chob-chini purified in milk.

3. *For the eyes*:—(*a*) Make a solution of tendrils of bittergourd in tender palmyra water through grinding and filtering, and use the clear solution for washing the eyes; (*b*) bundle crushed buds (or stalks?) of Mussænda Frondosa ("Vellila") and Indian Sarasaparilla ("Naru neendi") and squeeze drops into the eyes.

III. **Moar-chilanni.**—This group of Kapha-Masurika includes 5 sub-groups, one of which alone proves fatal. The presiding devatha is "Thulli-Marutha."

CLINICAL FEATURES.—1. General features are as in all Kapha-Masurikas (*vide* Section I). Additional features are:—All the sub-varieties have an 'incubation' period of 18 days (*i.e.*, the disease plays in the 'dhaatus' for 18 days' without the patient being aware of it). The invasion period is ushered in by rigours, high fever, intense headache, and unconsciousness; soon, appreciable swelling of body develops. In point of infectivity, Moar-chilanni group is on a par with Paaloori group.

2. Special diagnostic features:—(*a*) The urine of patient during the height of fever, if collected in a vessel and examined after a time, will be found to contain drops or particles ('Bindu')—of what? (*b*) A mixture of urine and gingelly oil, if kept and examined after a time, will appear as bubbles.

CLINICAL MANIFESTATIONS.—1. *Moar-chilanni proper*:—The rashes appear pepper-like, and with their eruptions, the fever and painful symptoms subside. The efflorescences become ripened on the tenth day, and then they emit the odour of pure buttermilk ("Moar"). When they dry up and break, the surfaces show a dark centre on a white base. The patient can be bathed on the 15th day.

2. *Paal-chilanni*:—The eruptions, when they appear, are pure white; and when they dry up and break, they leave surfaces whiter still. Their whiteness throughout has earned the name of Paal-chilanni for the disease ('Paal'=milk). The patient can be bathed on the tenth day.

3. *Kaal-chilanni*:—The rashes in this affection are few, and few and far between. When they break, the surfaces present the appearance of legs with toes. ('Kaal'=leg). The patient can be bathed on the 10th day.

4. *Thati-chilanni*:—The chief features here are swelling of the body, and the affection of eyes when the eruptions break or dehisce. Nothing is said about the termination of the affection.

5. *Vetti-chilanni*:—The initial symptoms and rashes are as in 'Moar-chilanni'. But, on the tenth day, the rashes break into fissures; and the patient develops brain symptoms of a raving type. He dies on the 18th day.

TREATMENT.—1. *Externally*: As indicated.

2. *Internally*:—(*a*) In the initial stage, decoction of Indian Sarasaparilla with sugar candy three times a day; (*b*) when fever has subsided and to help the rashes to desiccate, a decoction of root of Clitoria Ternalia, bael root, glycerizzæ, and coriander in equal parts is to be administered.

3. *For the eyes*:—(*a*) Crush and bundle Phyllanthus Niruri, onion, cumin seed in equal parts, soak in breast milk, and squeeze drops in the eyes; (*b*) mix lime-juice and the clear supernatant portion of sour gruel in equal parts, and use for washing the eyes.

IV. **Neer-chilanni.**—This holds 6 sub-groups, two of which prove fatal. The presiding devatha is "Neer-Marutha".

CLINICAL FEATURES.—1. General features are as in all Kapha-Masurikas. The following additional features are special to all varieties of Neer-chilanni. The 'incubation period' is 30 days, and the actual manifestation of the disease depends on the final pathological state of the body when the disturbed equilibrium manifestly affects the system and must show symptoms.

2. Special diagnostic features:—(*a*) Mixture of patient's urine, rice-washing-water and goat's milk will settle themselves in the vessel in separate layers; (*b*) betel-leaf, on one side of which patient's urine has been brushed, will show on the other side eruption-like projections; (*c*) application of paste of tortoise-shell in cow's milk to the face will bring out rashes on the chest, and application of the same to the chest will bring out rashes on the face—during the prodromal period, in either case.

CLINICAL MANIFESTATIONS.—1. *Neer-chilanni proper*:—"The period of invasion" is ushered in by high fever, cephalalgia, loss of consciousness, vomiting and diarrhœa. The course and prognosis: Cases with vomiting and diarrhœa at onset are of a mild type, the patient recovering and becoming fit for bath on the 18th day. On the other hand, the patient dies on the 22nd or 28th day if the following troubles appear *viz.*, exceeding foul smell about the patient, abdominal or throat pain, intensely red eyes, obstruction to voice, anxious gaze or stare of extreme fear, hallucination of "non-existent" calls and actions suited to follow the "caller", profuse sweats, terminal diarrhœa, feeling of oppressive and œdematous swellings over bregma or navel ("Moola-aadhaara"), and like ominous symptoms. Even the mild cases, that, under proper care, can be bathed on the 18th day, will swing between life and death for 48½ days, if not properly cared for.

The rashes, when they appear, are of the shape of 'Kottaikka' (?); and they come out on the third day. Though they may

seem to contain fluid, the contents are viscid; and when they rupture, necrotic cores will be exposed; and when the cores get removed and the wounds heal, the scars are severe black. Mature efflorescences, before rupture, will resemble Jambul fruits and emit mal-odour.

2. *Koor-chilanni*:—'Koor' = sharp or pointed. The general symptoms are as in and common to all varieties of Neer-chilanni. The rashes are discrete and particularly pointed; they become ripe and dry up in due course. Patient can be bathed on the 10th day.

3. *Pon-chilanni*:—This should not be confused with 'Pon-chilanni' and its branches below. The chief distinguishing feature of Pon-chilanni of Neer-chilanni group is that the rashes are tense with fluid; and the rashes are abundant. The patient can be bathed on the 12th day.

4. *Aar-chilanni*:—'Aaru'=six. The rashes appear on the 3rd day as usual; and on the fourth day of their appearance, *i.e.* the sixth ('Aaru') day of fever, they mature and become ripe fully. Because they become fully ripe, exactly on the 6th day, the affection is called Aar-chilanni. When they mature, they impart a special brilliance and shininess to the body. The patient can be bathed after $14^1/_2$ days.

5. *Poar-chilanni*:—'Poar'=battle. The rashes here appear in coloumns and resemble 'a body of troops' drawn up in deep files; hence, the name 'Poar-chilanni'. When the efflorescences are grown and have become filled, the intervening spaces present an ashy-grey hue. The patient can be bathed on the 10th day.

6. *Veer-chilanni*:—'Veerkal'=a swollen condition. Sand-like rashes appear all over the body; and with that, the body gets swollen. They recede some time later, and then there occurs bleeding per rectum. The patient dies on the 14th day.

TREATMENT.—1. *Externally*:—As indicated.

2. *Internally*:—(*a*) Decoction of equal parts of red sandal, cumin seeds and leaf-buds of horse-radish (Drum-stick plant) is to be given with sugarcandy added: (*b*) decoction of onions, cumin seeds, pavonia odorota and leaf-buds of cassia fistula in equal parts; this brings out the rashes in bold relief.

3. *For the eyes*:—Rub gold and silver in breast-milk and use the solution for washing the eyes.

V. **Pon-Chilanni.**—This affection holds 5 sub-groups, one of which proves fatal and another is of doubtful prognosis. The disease is called Pon-chilanni because of the golden colour of rashes when they ripen. Pon-chilanni is considered a lucky affection, affecting only the lucky. The patients who recover from the illness, it seems, are empowered and helped to acquire the wealth of 'Kubhera'. Pon-chilanni of Kapha-Masurika in many a way resembles

Kanaka-kkalloori of Piththa-Masurika. Presiding devatha is Soorga-Marutha.

CLINICAL FEATURES.—1. General features are as in Jwara of Kapha-Masurika (Section I). The additional features—common to all kinds of Pon-chilanni—are silk-red colour of the whole body, severe constipation in some cases, and giddiness accompained by nausea and blurred vision on getting up. Odour of edible figs when rashes ripen is a good sign.

2. Special diagnostic features :—(*a*) Patient's urine, if mixed with fowl's blood, will turn the colour of latter to white, (*b*) Patient's hair, if soaked in cow's milk and examined after a time, will be found to have turned grey.

CLINICAL MANIFESTATIONS.—1. *Pon-chilanni proper* :—The rashes appear on the third day like gunja-seeds in size and shape. With the appearance of rashes, the eyes begin to clear. When the rashes are ripe, they are golden in colour; they contain no pus, but a viscid substance. They usually desiccate and break; and on doing so, impart a shiny appearance to the body. If the patient is fortunate and does not die within 18 days, he can be bathed after 18 days.

2. *Mani-chilanni* :—The rashes resemble white beads and are discrete. They are streaked and contain no pus ; they desiccate and break. Patient can be bathed after 14 days. [*Cf.* 'Manian' under 'Kalloori' in Piththa-Masurika].

3. *Maanikka-chilanni* :—(' Maanikkam '=ruby). The rashes here resemble the fruits of the plant Jungle Geranium, and in course of time desiccate and break. Patient can be bathed on the 10th day.

4. *Aani-chilanni* :—' Aani ' = nail or peg. The rashes, when they appear, are nail-pointed ; on the 5th day of their appearance, they get flattened, globular, and umbilicated. When, after ripe, they rupture, firm necrotic cores become visible, and much blood and pus escape. Secondary (septic ?) rashes of different sizes and shapes appear in the neighbourhood of initial rashes, and these secondary rashes recede after a time. The patient dies on or after the 16th day.

5. *Kata-chilanni* :—The rashes are very dark and contain fluid. After a time since they break and desiccate, the patient can be bathed.

TREATMENT :—1. *Externally* :—As indicated.

2. *Internally* :—(*a*) Administer a mixture of fecula of Ficus Glomerata and onion in tender palmyra water. (*b*) Decoction of coriander and red sandal in equal parts. (*c*) Make a solution of grapes in pure cold water and give with candy for 3 days.

3. *For the eyes* :—Make a solution of coriander in tender cocoanut water, filter, and use the clear solution for washing the eyes; (*a*) Crush and bundle 'Amal-pori' (Mongoose plant) and cumin seeds, and squeeze drops into eyes three times a day.

VI. **Oatu-Chilanni.**—This affection is the least dangerous of Kapha-Masurikas; and Kapha-Masurikas being the least dangerous among the main varieties of Masurikas, Oatu-chilanni group may be considered as the least fatal type of "small-pox." But the fact should not be lost sight of that the rather mild affection may, under congenial circumstances, develop into the most dangerous and positively fatal forms. Besides, the tendency to infect is highest in this affection; being so, the attendants and visitors, liable to be lulled into a false sense of security, should be warned of the maximum infectivity of the affection. The presiding devatha is "Chandra-Marutha.

CLINICAL FEATURES:—General features are as in Jwara of Kapha-Masurikas (Sec. I). Special features are diarrhœa at onset and comparatively less congestion of eyes.

Special diagnostic features:—(*a*) During the height of fever, Chameli flowers, if scented, will induce vomiting; (*b*) application of Gorochan to root of ears will bring out rashes there, during prodromal period; likewise, application of charcoal from crematory as paste to glabella will bring out rashes there, during the prodromal period.

CLINICAL MANIFESTATIONS:—1. *Oatu-chilanni proper*: Rashes appear on the 3rd day; and simultaneously with that, there develop also "Kitakka-marungu" (bed-sores ?) They ripen within 7 days of their appearance; and when ripe, they exhibit the colour and odour of "Managola" (Realgar ?). The patient can be bathed after 10 days.

2. *Ul-chilanni*:—'Ul'=within. The rashes, a brief time after they come out, get fissured, discharging blood. The eyes are affected, and the patient dies on the 12th day. The affection is rare.

3. *Kattu-chilanni*:—The eruptions are black in colour, the affection runs a mild course, and the patient can be bathed after 10 days.

4. *Kaal-chilanni*:—The viscid rashes ripen from within outwards, break and desiccate. The patient can be bathed after 12 days. (*Vide* a similar affection under 'Moar-chilanni' in this Section).

5. *Maatu-chilanni*:—The efflorescences give the appearance of flies ('Makshika') sticking to the body; when they ripen, they become white or whitish. The patient can be bathed after 11 days. It should be noted that development of secondary boils over joints is a possible sequela in this affection.

TREATMENT:—1. *Externally*: As indicated.

2. *Internally*:—(*a*) Administer laaja powder mixed with the kernel of very tender cocoanut three times a day. (*b*) Rub Strychnos Potatorum in tender cocoanut water, boil and "digest" the whole, and administer. (*c*) Administer a decoction of onions, leaves of Krishna Tulsi, Strychnos Potatorum and 'Makki-poo.'

3. *For the eyes*:—Make a solution of purified Cowries in decoction of cumin seeds, and use for washing the eyes. (2) Crush and bundle 'Kutangal' (?) and onions in equal parts, and squeeze drops into the eyes.

Some Notes on Kapha-Masurikas

1. Kapha-Masurikas are certainly the least dangerous among the three main groups of Masurikas. While 27 out of 32 varieties of Vaata-Masurikas invariably prove fatal and 12 out of 32 do so among Piththa-Masurikas, only 9 out of 32 varieties of Kapha-Masurikas end in certain death of the patient; 18 varieties of the last have a good chance of recovery, while the remaining 5 have a fair chance of getting round. Of the cases that prove fatal, those of Ninal-paaloori and Veer-chilanni die of internal hæmorrhage and recession of rashes; those of Kootta-chilanni, Aani-chilanni and Ul-chilanni die of external bleeding into eruptions when the latter break; those of Vitta-chilanni and Vetti-chilanni meet their ends through raving delirium and exciting brain complications; cases of Ven-chilanni proper prove fatal through septic absorption and severe toxæmia; and those of Thati-chilanni die of "un-mentioned" causes. It will be observed that of the 9 fatal varieties 5 prove fatal through internal or external bleeding—*i.e.*, more than 50%.

2. Kapha-Masurikas, though less fatal, are more infectious. In fact, it would appear that infectivity runs per contra to fatality among Masurikas—the less fatal varieties proving more infectious and *vice versa*.

3. The facts of minimum fatality and maximum infectivity and the facts that in non-fatal cases the patients can be bathed within a fortnight of the onset of disease (many of them, even as early on the 10th day)—these facts make one suspect that cases of Kapha-Masurika (at least, cases that are mild and do recover) are really cases of Varicella and not cases of Variola; and perhaps the rows of rashes in Poar-chilanni of Neer-chilanni group and the 'bed-sores' in Oatu-chilanni proper bespeak of herpetic eruptions commonly associated with chicken-pox. We shall revert to the subject in the next and concluding section where we shall, in brief, make a comparative study of Masurikas of old and exanthemata of new.

(To be continued).

Atropine—Antidote to Phosphorus Poisoning

The newer phosphorus containing insecticides, are certainly poisonous and when poisoning occurs as a result of excessive and careless handling, the early signs observed are tightness of the chest, slight twitching of the muscles of the eyelids and tongue and contracted pupils. Dr. Goldblatt recommends, for the earlier stages, a dose of $\frac{1}{4}$ mg. of Atropine Sulphate by mouth. If symptoms are not relieved in an hour, repeat the dose, observing the patient closely. If he does not improve but gets worse, 1 mg. of Atropine Sulphate may be injected subcutaneously or intravenously. For later stages of poisoning, even higher doses may be given.

The organic insecticides which include hexaethyltetra phosphate, and tetra ethyl pyrophosphate and related products exert an effect comparable to that of acetylcholine.—(*Pharmac. Jour.*, March 1950).

INTERPRETATION OF LABORATORY FINDINGS AS AN AID TO DIAGNOSIS*

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Fæces—Its diagnostic importance

MUCH information can be obtained by mere inspection of fæces and the most important one can be gathered from its character. The normal character should always be borne in mind. It is formed, soft with light or dark brown or brownish yellow colour, with slight offensive odour. Typical characteristic stools as described in diseases are not always found, for example, in cases of acute amœbic dysentery we do not get the characteristic findings early, though at later stages in most cases we get it, but in subacute or chronic cases the character varies. It may be scanty, grayish green to reddish brown and mucoid. The stool in other cases may be entirely of blood and mucus, pure or mixed up with a little fæcal matter, or blood-stained purulent material with almost always of characteristic fœtid odour. Sometimes stools may be of liquid, yellow with only flakes of mucus, or it may be of slimy nature.

In cases of acute bacillary dysentery which is generally caused by shigella—the typical stool appears to be liquid watery in which flakes of mucus float—the liquid portion may be of red colour denoting blood or the flakes may be blood-streaked only. Here we get the colour of the blood as bright red in contrast with amœbic where it is blackish red. The odour here is not characteristic, rather it can be said inodorous.

In cases of flagellate protozoal infection of which giardia infection (scientifically it is non-pathogenic) may be taken as an example, stool almost always is liquid yellow with little mucus, bulky and often frothy and oily—the reason possibly is due to the fact that the habitat of giardia is in the duodenum where the proper digestion is hampered. One of the characteristic points of this infection is the occurrence of alternate diarrhœa and constipation.

In cases of ciliate protozoal infection (type–Balantidium coli) the appearance of stool is almost like that of an acute attack of bacillary dysentery. In cases of other infection of intestinal tract (cholera), after some period of the onset characteristic rice-water stool as a result of absence of bile secretion gives one the definite point to arrive at a diagnosis.

The other enteric group of organism of which E. typhosum is important the character varies very much—from pea soup colour of stool to frank blood as a result of perforation of ulcer. Often it may be found as greenish liquid with masses of mucus.

Coccal infection may cause diarrhœa and sometimes the appearance of stool is liquid red, but presence of R.B.Cs. may not be revealed by microscopic examination. In all probability such a type is due to hæmolytic strain (Streptococcus).

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* Specially contributed to THE ANTISEPTIC.

The blood-inhabiting protozoa (plasmodia and leishmania-donovani) may cause diarrhœa, the former may often show of choleric nature or even like that of acute dysentery; in the latter, the diarrhœa shows of small quantity of various colours—the number of stools become great and this is almost always terminal.

In deficiency disease and anæmia of macrocytic type, the character is liquid mixed up with fæcal matter or practically of watery nature, odour is offensive or sour and stools are bulky pointing to indigestion.

The appearance in diseases of liver is almost always grayish or muddy, frothy and bulky with offensive odour (cause—absence of bile).

In sprue or para-sprue, we expect to get frothy and bulky stools with steatorrhœa.

In ulcerative colitis, blood-mixed stool with mucus is the ordinary feature, but may be only of stools with mucus.

In infantile diarrhœa, if caused by dentition, it is at first simply yellow liquid with scanty fæcal matter leading on to greenish with strips of mucus and watery and then to simply watery and mucus (all possibly due to want of proper nutrition—inanition).

Climatic and altitudinal diarrhœas are simple ones–the quantity is large, liquid or semi-liquid in consistency containing fæcal matter and motions passed with sounds.

Helminthic diarrhœa shows itself to be mucoid, yellowish, semi-solid, often in many cases with streaks of blood, but the infection caused by ankylostoma often shows stools of blackish colour due to altered blood.

Flattened ribbon-like stools result from some obstruction in the rectum, such as from tumour or stricture caused by a healed ulcer—may be of syphilitic origin. If such a stool shows streaks of blood, it gives a suspicion for carcinoma in the absence of bleeding piles.

Mucous present in stools signifies irritation or inflammation of the intestine. If mucous is small and intimately mixed up with stools, the trouble is probably in small intestine. Wholly mucus and blood-stained stools point to dysentery, ileo-colitis, intussusception, mucus colitis or membranous enteritis or secretory neuroses. Shreds or ribbons of altered mucus representing complete casts of portions of bowels are passed especially after an enema, and after washing such a stool segmented strands may appear to delude to observer suggesting fragments of tape worm. By seiving stools, concentrations such as gallstones by their faceted surface can be recognised.

Odour of stool is another important finding. Stools emitting a very foul stench odour are suggestive of malignant or syphilitic or ulceration of rectum or gangrenous condition due to any causes. As previously noted in acute bacillary dysentery stools give no characteristic odour.

Reaction of fæces to litmus is important for diagnostic purpose. In almost all cases the reaction is acid. In cholera,

bacillary dysentery and even in pure and simple diarrhœa and in many other cases where the passing of intestinal contents be very hurried, the stools give reaction to alkalinity—sometimes in cases of acute amœbic dysentery, the reaction of stools become alkaline, the reason of course being due to excess of blood.

Other chemical tests also play an important role to aid clinical diagnosis. Amount of blood in stools, too small to detect by simple inspection, constitute occult blood, and this requires a chemical test for detection.

What does the blood in stools—naked eye appearance or detection by chemical test in case of occult blood—denote, is a potential question. The blood in stool may come from anywhere of the long alimentary tract—bleeding gums, ulcers in the tract, bleeding piles, dysentery, etc. Blood may also be of lung origin—hæmoptysis—in which case the blood may get into stomach by regurgitation (act of deglutition). By only getting the occult blood test positive, one is not justified to lean to make a diagnosis of duodenal ulcer. Repeated tests are required and if in seven successive days the stools show positive reaction in different times, then and then only the case should be looked upon with suspicion and this is the consensus of opinion. If the colour of blood is bright red, one can assume that its origin is at rectum or a little higher up. If colour is blackish and pure blood, the origin is venous and may be taken as of coming from upper bowels. If the colour shows tarry or black character, certainly the blood is coming from higher up the alimentary tract. The colour-change is due to the blood being digested with formation of hæmatin. Blackness of stool due to blood may be distinguished from that produced by drugs by a simple physical test. "Mix thoroughly stool with twice its volume of water and allow to stand. The supernatant fluid is tinged and becomes reddish in case of blood, and which in other conditions remains dark or greenish", but one is to remember that dyes such as hæmotoxylene or any fruit with red colour, if administered to patients, or people who are heavy consumers of beetle, may pass reddish stools and that may impart the red tinge to the supernatant fluid in the above physical test mentioned and the chemical test therefore is of so much importance here. It is well to bear in mind that various drugs, etc., may give rise to various colours, *e.g.* cocoa and chocolate impart dark gray colour, calomel—green due to biliverdin, Iron and Bismuth—dark brown or black ; hæmatoxylene—red. In this connection one must not forget the frank blood passed along with stools in bleeding piles, but here we get pure blood which generally comes by drops earlier to passing of stools.

In cases of deviation from normal colour of stools, one may carry out the test for bile, the absence of which will point towards deficiency of liver action. Green stools in children, if accompanied by symptoms, is pathological, such as in cases of green diarrhœa—the colour is due to biliverdin. Chromogenic bacterial infection

may also impart such or other colour. Milk diet in excess gives a light yellow colour. Whitish curd-like masses in stools of infants is due to either imperfect digestion of fat or casein or to excess of these diets. When there is an interference of fat digestion, *e.g.* in pancreatic disease, stools appear greasy and consist of fat or its derivative in excess. An important fact is to be always borne in mind that after administration of fat, castor oil for example, the stools may show fat globules microscopically.

The tests for enzymes are carried out to find out, *e.g.*, the pancreatic deficiency, but authorities are divided into two camps as to the validity of the test. Any way the following facts may beneficially be remembered. Two ferments, amylase and trypsin, are normally present in fæces. Pancreatic disease or obstruction of pancreatic duct cause the ferments diminish or become absent. So a quantitative test and a qualitative as well may be helpful. Of these two ferments amylase is the most important since the action of trypsin may be simulated by Erepsin or the proteolytic activity of bacteria.

Microscopical Appearances

A good deal of information as to the nature of infection may be had from this—helminthic or protozoal or otherwise. Elucidation regarding dyspeptic condition, defect in liver action etc., may be recognized also, *e.g.*, starch granules, fatty acid crystals, neutral fat etc., point to particular deficiency. Infantile liver causes diarrhœa, but microscopic appearance does not give any clue by characteristic findings, *i.e.*, there is nothing which is characteristic. Presence of Charcot-Leydon crystals point to chronic amœbiasis mostly, but may be encountered in other cases as well, *e.g.*, helminthic infection (ankylostoma particularly). Fatty acid crystals, neutral fats signify deficiency in fat absorption and only be encountered in pancreatic diseases provided no bland oil was administered. Presence of cysts of pathogenic protozoa suggests that the disease has gone up to chronic stage and such cases need handling with great care. In bacillary type of dysentery—in acute cases—large number of macrophages are seen. If R.B.Cs. are present in stools, the character may be noted—if they are discrete, and microscopic field shows abundant in addition to other picture, it is generally of bacillary origin, but in amœbic cases the red cells show tendency to agglomeration. If microscopic findings show pus cells and epithelial cells mainly entangled in mucus the cases are generally of enteritis. Presence of A.F.B. in stools is less significant than in other materials as their presence in stools may be wholly due to swallowed infected sputum. Pure cholera stools do not ordinarily show pus cells, but smear examination reveals comma bacillus.

***Note*:—The examination of stool is not complete if at least ordinary bacteriological examination by way of culture be not undertaken.**

Special examination, *e.g.*, cholera red reaction or special cultural examination or animal inoculation, only gives many clues to facilitate diagnosis.

PHYSIOLOGICAL FACTS ABOUT MICTURITION*

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EXCRETORY system persists in us because we are still imperfect from the point of view of evolution. Were it not there, waste products would have accumulated and life could have extinguished. All that we eat or drink is not completely metabolised and transformed into energy, static or dynamic.

Mechanism of urine formation.—The urine which is voided by a healthy human being is quite different from what it is actually formed initially by the kidneys. The manufacture of urine is the sole proprietary right of the kidneys, but it does not mean that the kidneys have only this function to perform. They serve a more important function, rather a very vital function, of maintaining the normal pH of blood in the body, which is extremely essential for the optimal activity of the protoplasm.

Structure of the kidney:—The human kidney is phylogenetically the metanephros, the older pronephros and metonephros exist only during embryonic development of the fœtus. The kidney is a compound tubular organ having an external lining, called 'cortex', and an inner one, the 'medulla'. The functional unit of the kidney is a "nephron". A nephron has got 2 main parts.

(1) *Bowmann's capsule*:—It encloses a tuft of very thin-celled, blood capillaries, which are merely an intra-renal portion of the vast capillary bed. 'These tiny intra-renal tufts of capillaries enclosed in the Bowmann's capsule are called the 'glomeruli.'

(2) *Uriniferous tubule*:—The renal tubule in its entire length is lined by a single layer of cells; and the more we proceed towards the collecting tubule from the neck of the uriniferous tubule, the cells richer in their protoplasmic contents are found.

The length of the human renal-tubule is approximately $1^1/_6$", its diameter being about 44–60μ.

BLOOD SUPPLY.—Renal artery enters the kidney at the hilum, and there divides into a series of 11 branches that pass towards the renal cortex, termed as the "inter-lobular arterioles". These arterioles, later called as 'afferent vessels', ramify to constitute the glomerular tufts, and then turn back without anastomosing, to be called the 'efferent vessels.'

These efferent vessels, like a portal system in miniature, further break up into several minute capillaries to supply the uriniferous tubules, and finally unite to form the venules and then the renal vein.

Blood pressure in the glomerular tufts is much higher normally than it is in the capillaries elsewhere in the body. And, not all the

* Specially contributed to THE ANTISEPTIC.

glomeruli are working at any particular moment, as if there is also "shift" system of activity in the glomeruli.

Nerves have probably no direct influence on the renal activity except that they do modify the blood-supply to the kidneys through a humeral mechanism.

Functions of the kidneys.—Kidney in its excretory role is closely related to the skin, while in maintaining the pH of blood to the lungs.

1. *Bowmann's capsules*:—It has been experimentally proved that the Bowmann's capsules have no physiological activity. The filtration of urine by glomeruli is a simple physical process. Nature is a very economical engineer. Histological structure of the Bowmann's capsule consists of a single layer of nongranular cells deficient, rather, lacking practically, in protoplasm and its nuclei are inconspicuous. This structure itself is adapted to the action of filtration of urine, which is a non-vital process, and wherein the wall of the Bowmann's capsule acts as a daily sink membrane allowing the crystalloids to cross the "barrier" and retaining the more important constituents of the blood *viz.*, the colloids. That is, the urine poured by the glomeruli = plasma minus colloids. As filtration of urine is a physical process, a change in the blood pressure influences the amount of urine filtered out; but any alteration in the ostriotic pressure affects a little.

2. *Uriniferous tubules*:—The uriniferous tubules, on the contrary, have got their wall lined by a single layer of cells rich in protoplasm and with well-defined nuclei. This itself indicates that the uriniferous tubules are, by Nature, meant to execute a vital function. An abundant provision of protoplasm to the cells of the uriniferous tubules must have got some purpose to serve. And it is actually so. There is a sort of selective re-absorption capacity in the cells of the uriniferous tubules, a very complicated affair, meaning thereby that a further change in the filtered urine during its onward course in the uriniferous tubules involves a vital process. The "torrents of undifferentiated filtrate" which amounts to something like 150 litres in 24 hours (actual urine discharged by a healthy adult in 24 hours is approximately 1·5 litres)—such an enormous quantity which would necessitate not only an extraordinary burden to the kidneys but also an unwanted wastage of the threshold substances is definitely reduced and changed in quality in its passage down in uriniferous tubules. The useful substances are picked up and re-absorbed, whereas the things otherwise harmful to the human system are eliminated and excreted. Water, glucose and chlorides which are essential constituents of the blood, and are called as 'threshold substances' by Richards, are taken up from the filtrate. The inorganic threshold substances are absorbed along the distal tubule, while the organic threshold substances are absorbed from the proximal tubule; water is absorbed from the

entire length of the uriniferous tubule. Thus, it is seen that urine is the medium for only nonvolatile water-soluble solid wastes. A change in the blood pressure has practically no effect on the quality of the urine, but a change in the osmotic pressure does affect the quality of the physiological diffusion.

Other proofs :—There is yet another way to prove that Bowmann's capsule acts as a filter, while the uriniferous tubules act as dialysers. When two samples of blood are taken, one from the efferent vessels after its circulation in the glomerular tufts, and the other from the renal veins, and if these are examined for their individual contents of oxygen, it would be found that whereas in the former case oxygen content is the same as it is in the blood of the efferent vessels, in the latter case there is less oxygen content in the blood of the renal veins than in that of the efferent vessels. This clearly indicates that some of the oxygen is definitely used by the cells of the uriniferous tubules, whereas none by the cells of Bowmann's capsules, and " no vital activity is ever possible without the utilisation of oxygen " is the fundamental fact of physiology. Hence it is proved that the Bowmann's capsule acts passively, whereas the uriniferous tubules work actively ; the former acts as a mechanical agent, the latter as " living " dialyses. A kidney consumes as much as about 0·05 c.c. of oxygen per gram of its substance per minute.

Nature's economy.—Nature is never extravagant. Its economical working is well appreciated when we observe that the blood supply is first to the structure which little uses O_2, *i.e.*, to the Bowmann's capsules, and thence to the uriniferous tubules, which utilise oxygen.

Path of secretion of urine.—Kidney→renal pelvis→ureter →urinary bladder. The bladder is thus merely a storehouse of the manufactured urine, from where it is voided to the exterior from time to time.

Nerve supply to the urinary bladder.—Urinary bladder is innervated by the autonomic portion of the nervous system. The A.N.S. is distributed as:—

A.N.S.—
- P.N.S.→Causes relaxation of the sphincters, and the contraction of the bladder wall—hence urine passed out.
- S.N.S.→Causes contraction of the sphincters, and the relaxation of the bladder wall—hence urine withheld back.

Sympathetic nerve supply is from the hypogastric nerves, while the parasympathetic one is *via* the nervi erigentes. Lower centre is in the lumbar enlargement, and the higher centre in the hypothalamas. A new third centre is created in the motor cortex of the cerebrum when a voluntary check over the act is cultivated.

The fundamental facts about micturition and its physiology are thus outlined above. Principally, micturition is an unconditioned reflex for which a gradual rise in the intra-vesical pressure of the urine and its reaching to a certain optimum is the all important exciting stimulus for the execution of the act.

The sensation of distension is the sole basic phenomenon for the discharge of voluntary micturition, and this sensation is perceived when the tension of urine in the bladder reaches about 15-18 c.m. of water.

Summary.—1. Excretory system persists in us during the course of evolution as we are still imperfect.

2. Manufacture of urine is the sole proprietary right of the kidneys, although their more important function is to maintain the optimum pH of blood.

3. Nature is a very economical engineer, and this we can observe if we study the histology and circulation of the Bowmann's capsule and of the uriniferous tubules.

4. Bowmann's capsules are merely mechanical filters involving no use of oxygen, while the uriniferous tubules are physiological dialyses involving the use of oxygen.

5. Fundamentally, micturition is an unconditioned reflex requiring a harmonious co-operation of the two components of the autonomic nervous system, and for the execution of micturition, a certain optimum intravesical tension of urine is most important.

Comparative Effects of Water, Soda-water, Sod. Chloride and Soda Bicarb Solutions on Gastric Secretion

From time to time one sees or hears the statement that carbonated soda-water, or soda bicarb solution stimulates gastric secretion in men. Hale *et al* of the College of Medicine, Chicago, carried out controlled tests to determine whether soda-water or soda bicarbonate had a significantly different effect from an equal quantity of plain water or normal saline on the secretion of acid by the stomach in human subjects. The subjects were normal young adult males and male adults in whom the diagnosis of duodenal ulcer had been radiologically established. The results showed—

(1) In normal human subjects (*a*) carbonated soda-water does not stimulate gastric secretion or affect gastric emptying to any greater extent than plain water; (*b*) normal saline and normal soda bicarb solutions leave the stomach at about the same rate.

(2) In cases of duodenal ulcer, normal soda bicarb solution leaves the stomach more rapidly than normal saline solution.

(3) In contrast to plain (distilled) water, normal saline does not stimulate gastric acid secretion.—(*Jour. Lab. Clin. Med.*, 35, 2, pp. 249-252, 1950).

SYMPTOMS OF 'ACUTE ABDOMEN' IN CHOLERA

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IN over ten years of my practice I have treated a comparatively larger number of cholera cases. I was very recently faced with such symptoms of "acute abdomen" as to advise the removal of the patient to Patna for operation. This, of course, I did in consultation with one of the most senior members of the profession who agreed with my diagnosis which proved, in the end, to be utterly wrong, though the treatment which I had given was credited with success.

With the idea of confessing my shortcomings and placing this queer complicating symptomatology of a cholera case before the profession, I propose to give an account of the same.

On 30–8–'50 at 9 p.m. I was called to see one Jaidayal Singh, aged about 50 years, for the treatment of diarrhœa and vomiting. The patient complained of—

(1) 7–8 watery motions since the morning. Vomiting had occurred 3–4 times. The first vomit consisted mainly of undigested food matter which the patient had taken the previous day; (2) extreme weakness; (3) violent thirst; and (4) restlessness.

On examination the patient revealed the following.—(1) Constitutionally thin and anæmic; (2) no sign of dehydration except slight sinking of the eyeballs which I attributed to his build; (3) tongue—moist, coated with white fur; (4) pulse—110 p.m. regular, V & T low; (5) temperature 97°F (armpit); (6) no abnormality in heart and lungs; (7) abdomen—soft and gurgling, spleen and liver not enlarged; and (8) urinary system—urination occurred with motions. Bladder empty.

History:—The patient had his meal very late on the previous day after great exertion which was unusual for him. A few days back there had been two cases of cholera, of whom one died in the vicinity.

TREATMENT.—1. Sulfaguanidine 8 tablets plus 4 tablets Sodamint in ½ ch. of water was given stat and half the dose was recommended 3 hourly.

2. Grape sugar solution 50 c.c. 25%—2 c.c. Cycliton was given I.V. slowly.

Result:—Pulse improved, thirst and restlessness decreased.

I left the patient at 10 p.m. with the following instructions:—(1) to take the medicines as directed; (2) to take sips of glucose water frequently; (3) to pass his urine and stool in bed; and

(4) to send for me immediately if weakness or restlessness increased or cramps appeared.

The same night at 1-30 a.m., a man knocked at my door and reported that the patient's condition had worsened and I was to go there with Saline arrangements.

I rushed immediately with necessary arrangements and found that the patient was complaining of:—(1) Extreme prostration; (2) cramps were evident in legs and abdomen; and (3) anuria and restlessness.

On examination.—(1) Facies of cholera established, husky voice evident; (2) signs of dehydration with sinking of eyeballs and shrivelling of fingers and toes were evident; (3) pulse quick and thready; and (4) axillary temperature subnormal; (5) heart sounds feeble; and (6) bladder not distended.

I asked my assistant to prepare two pints of Normal Saline 4% Glucose and in the meantime I injected 50 c.c. 25% Glucose and 2 c.c. Cycliton I.V.

The pulse returned to normal after one pint of Saline transfusion and the patient began to feel much better. However, I thought it better to transfuse another pint of Saline and Glucose and added $^1/_2$ c.c. Adrenalin and gr 1/100 Atropine into that. By the time 16 oz. of next pint of Saline was finished, the patient complained of sudden pain in the abdomen on which I withdrew the saline needle from his vein and switched it on to his armpit subcutaneously. By this time the pain had increased all the more inspite of his feeling well otherwise. I kept waiting and watching by his side till 4 a. m., but the pain was not reduced. One tab of "Cibalgin" was given stat and I left the patient when there was some amelioration. The abdomen was not exhibiting any particular sign till then and I attributed the pain to be due to intestinal spasms although it is rare in cholera. Acute dysentery of course was a probability.

I had hardly a wink of sleep when a man came and awakened me at 6 a. m. on 31-8-'50 and reported that the patient's condition had worsened still. On my arrival I found that the patient was—(1) almost pulseless and restless; (2) complained of severe pain in the abdomen which had become board-like rigid. The thighs were in flexion. On percussion it was tympanitic all over except on the lying side. The liver dullness had disappeared. There was no bowel sound; and (3) vomiting and diarrhœa had stopped since the beginning of pain.

On the above findings I suspected gastric perforation, especially when I was able to elicit the history of previous pains relieved with Soda. I called in one of my senior colleagues for consultation and he corroborated my diagnosis and the question of removing the patient to Patna for operation was considered. As he was almost pulseless (primary shock ?), it was thought better to wait till it improved. It was decided to push another pint of Saline, so in the meantime

50 c.c. of 25% Glucose—2 c.c. Cycliton was again given I.V. by the time Saline was ready. But there was no improvement until one pint of Saline was transfused.

The patient was removed to Patna on a country boat at 11 a.m. on the same day with a history and treatment note prepared by me.

Discussion.—**A.** Was this a case of cholera or acute gastro-enteritis culminating into intra abdominal perforation ?

POINTS OF INTEREST:—*Cholera*:—(1) History of cholera in the vicinity; (2) signs of dehydration; (3) no nausea or griping; (4) toxæmia lesser; and (5) vomits profuse.

Acute gastroenteritis:—(1) Acute pain in the abdomen; (2) vomiting and dirrhœa; and (3) rigidity of abdomen.

More points were in favour of cholera.

B. Can acute addomen supervene in a case as above ?

(1) Gastric perforation can certainly occur ina case of incipient ulcer due to lowered vitality, starvation and sudden accumulation of a large amount of fluid in the stomach; (2) rupture of some intra-abdominal arteriosclerotic vessel might possibly occur in an aged person due to rapid Saline transfusion; (3) paralytic ilius may occur in a case of cholera but in this condition there is no pain; and (4) acute uræmia sometimes presents the picture of acute abdomen but the pathology of pain is not explainable.

In the above case we removed the patient in favour of more serious condition about which we were more confident and for which we had nothing under our domain to deal with. Our conviction was much in favour of No. A or No. B 2.

I had no information of the patient until the next morning although I was very eager and anxious to know about the progress of the case and confirmation of the diagnosis.

At about 10-30 a.m. on 1-9-'50 one of the attendants returned from Patna with a smiling face and informed me that the patient was doing well. He said that the pain began to decrease while he was on the way and completely subsided when the patient had a motion when he reached there, his pulse maintained although no treatment except Alkaline mixture, cholera powder and an injection of "Carbachol was given after his admission in the Cholera Ward of the P.W.M. College Hospital, Patna. The patient was discharged from the hospital and came back on 4-9-'50. I had the opportunity of examining him again after his arrival when he was free of troubles except some residual weakness.

Probably this case developed toxic uræmia which presented the symptoms of acute abdomen, or there had been some small gastric leakage which may have sealed automatically. But in the former case, the pathology of pain remains a problem and requires elucidation by those competent to explain.

INTRAVENOUS INJECTION OF UREA STIBAMINE AND NITRITOID CRISIS

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ACUTE vasoparetic reaction or nitritoid crisis is the term employed by Milton because the symptoms and lesions resemble inhalations of "Amyl Nitrite". These reactions are produced by the intravenous injection of many substances such as Urea Stibamine, N.A.B., Glucose etc., and are common among individuals with Adrenalin insufficiency and thyroid disturbances. They have been attributed to the disturbance of the "sympathetic system". But it is more likely that the toxicity of the drug is responsible for the manifestation of the acute symptoms, or they may be due to some abnormal changes in the capillaries, or the combination of both (Chopra).

The symptoms manifested shortly or immediately after and rarely during the injection are the flushing of the face, nausea, vomiting, abdominal pain and sometimes diarrhœa and collapse. There is sudden fall of blood pressure with feeble, small and imperceptible pulse ; constriction of the throat, swelling of the lips and respiratory embarrassment are also present. Seldom there is loss of consciousness which may last for several hours and the case may either revive or end fatally. Reactionary fever is always present and comes on not at once but a little later. These reactions though alarming are rarely fatal.

I met with only one case of fatal reaction during my whole period of service of about 30 years. I came across cases with very severe reactions and they all revived after proper treatment.

These reactions are also called "Anaphylactoid reaction", because they resemble, "Anaphylactic shock", which is due to antigen anti-body reaction. These are quite different from "serum sickness ". In serum sickness the symptoms come on 8 to 12 days after the injection and are characterised by erythema, urticarial rash, joint pain, and fever, and are more frequent after intramuscular than after intravenous injection. Anaphylactic reaction according to Park is 1 in 20,000 and fatality 1 in 50,000. I presume reactions after intravenous injection of Urea Stibamine are much higher, though the fatality figure may not exceed that due to anaphylactic reaction.

I had the misfortune to find fatal reaction in a case receiving only ·05 gm. of Urea Stibamine (Union Drug & Co.) in the first injection. This was an early case of kala-azar diagnosed after usual serum test, his general health being not much affected. I gave him ·05 gr. Urea Stibamine intravenously. Just after the injection I noticed that he was reeling and suspecting something wrong. I at

once made him lie down on the floor. His eyes became red and he was restless. He could hardly speak and his voice was husky. He expressed his agony by the gesture of his hands. Pulse was very feeble and almost imperceptible. Soon he became cyanosed and had hurried and stertorous breathing, followed by complete loss of consciousness and death. Specific treatment of the condition with Adrenalin Chloride could not be given to the patient, it being then out of dispensary stock. All cardiac stimulants like Digitalin, Strychnine, Ether Camphor were given parenterally but with no response. Artificial respiration and cardiac massage were also resorted to but all in vain and the patient died within 10 to 15 minutes after injection. I never came across deep cyanosis in any of my previous cases with very severe reactions and never ending fatally, though such cases do occur in very rare instances.

I think it will not be out of place to cite here a case of snake-bite treated with intravenous injection of "Anti-venom" (Pasteur Institute, Kausali). Within a few seconds after the injection the patient suddenly turned deep blue and died immediately after. I then attributed this deep cyanosis to a sudden change in the blood due to the sudden dislodgement of snake venom into the circulation after the intravenous injection. But now I am led to believe that the deep cyanosis and death were due not to snake venom but to anaphylactic shock which resulted from the intravenous injection of "anti-venom" and as a result of antigen anti-body reaction.

The cause of, "Nitritoid crisis", after intravenous injection, has not been accurately found out, different authors holding different views.

1. Col. CHOPRA :—His contention is that some change—precipitation—takes place in the blood as soon as the solution reaches the circulation. Precipitation is often seen to take place within the syringe during the injection when blood enters the barrel of the syringe. There is no aggutination and hæmolysis.

It is not understood why reaction generally does not take place in the first injection and in every injection inspite of the precipitation of the blood being present in all cases but occurs late when the course of treatment is about to be completed. There is no denying the fact that precipitation is less when the patient is about to be cured and when he is in the last phase of treatment. Hence his argument does not stand.

2. Dr. U. N. BRAHMACHARI :—According to Dr. Brahmachari reactions are due to, "Idiosyncracy" or "over-susceptibility". It is true, certain individuals are hypersensitive to certain drugs such as Quinine, Mercury, Pot. Iodide etc. But hypersensitive persons are always susceptible to the first dose and also to all subsequent doses if not de-sensitised. After intravenous injection, reaction generally takes place not in the first injection but late in the 5th or 6th injection or towards the completion of the course.

Thus the explanation is vague and not based on strong grounds.

3. Cumulative action :—Some say this might be the cause.

As elimination of Antimony Salts (Urea Stibamine in the present case) after intravenous injection is very rapid and because injections are generally repeated every second or third day, the chance of accumulation of the drug is almost nil. Besides, there can be no accumulation in the first injection as in the present case. Hence the argument is groundless.

4. High toxic doses may produce reactions. But in the present case the dose is not sufficiently high to produce reactions. Again, in all cases of reactions, the doses were not proved to be high to produce symptoms of reactions. Hence the cause of reaction must be something else.

5. Severe reactions following intravenous injections may be due, at least, in part to the fact that the pH of the fluid introduced is decidedly more acid or alkaline than that of the blood. Such solutions should be buffered. When not done so *i.e.*, when not buffered, reaction may occur. But the fact that reaction takes place after several injections and not in the first, does not lend support to the above contention.

Buffers.—If acid or alkali be added to a solution of a strong base or acid it will be found that usually the pH is markedly affected. Certain substances, however, when present in the solution, act to modify the usual effect in such a manner that the change in the pH may be practically inappreciable. Such substances are known as buffers. The important buffers are Bi-carbonates, Proteins (specially Hæmoglobin) and Phosphates.

Blood contains substances like Acid and Alkaline Phosphates and Sodium Bicarbonate which together with Hæmoglobin and Protein act as a powerful buffer and prevent reaction of the blood being easily changed.

Note :—A few days after the incident, it came to my knowledge that the patient in question kept on starvation for two or three days before the injection owing to domestic quarrel.

Starvation produces acidosis, *i.e.*, changes the pH of blood to the acid side. As long as pH is normal (7·3 to 7·5), the acid base equilibrium is normal or compensated, otherwise it is uncompensated and life is threatened. It is possible that the change of pH to the acid side due to starvation might have something to do with the death of the patient above alluded to. Again, as the reaction came on in the first injection and with a small and non-lethal dose it is probable that the death was due to "Hypersensitiveness."

It is essential that the cause of reaction, after intravenous injection, so alarming and sometimes fatal, should be found out.

A CLINICAL OBSERVATION OF PHTHALYL SULPHACETAMIDE IN B. DYSENTERY

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SINCE the reference to the above mentioned drug in Jan '51 issue of THE ANTISEPTIC by U. P. Basu and A. N. Bose of the Bengal Immunity Research Institute, Calcutta, this clinic gave a fair trial to this drug in a series of 12 B. dysentery cases. The use of this drug was confined to patients whose ages ranged between one month to 7 years. The drug was used to treat more than 12 cases of dysentery but the clinical symptoms of B. dysentery were very clear in only 12 cases.

All these 12 cases showed more or less the same symptoms *e.g.* fever at the onset, gripping pain in the abdomen with number of loose motions loaded with blood and mucous. Eight of them showed an additional symptom of vomiting everything out. Two were complicated by the native *vaidians* in as much as their bowels were constipated which added more distress to the already toxic patient.

The drug in the form of ·5 gm. tablets is known by its trade name as Enterocid (P-Phthalyl Amino Benzene Sulphonacetamide). The drug was administered in the following dosage:—

3 months to 1 year—½ tab. q. 4 h for 2 days, q. i. d. for 2 days.
1 year to 3 years—½ tab. q. 4 h for 2 days, q. i. d. for 2 days.
4 years to 7 years—1 tab. q. 4 h for 1 day, ½ tab. q. i. d. for 3 days.

For a complete cure a 4 days' treatment was quite sufficient.

The required amount of the drug was given in the form of a mixture, added to each dose 3 to 10 grs. of Sodi-Citrate and sweetened by any simple syrup. Children take this mixture very easily. In cases of vomiting, if the first dose is vomited out, immediately the next dose is given and this is well tolerated. If the child retains one dose, the improvement will be seen within the next 2 to 4 hours' time. Glucose water by mouth in small teaspoonful doses, is an added advantage. The sucking babies were not disallowed milk but the older ones used to rice etc., were restricted to fluids alone.

It is stated that the drug is also useful in the treatment of ulcerative colitis, typhoid fever, cholera and in intestinal surgery. The drug is not yet freely available in the market but we hope that it will be given a fair trial in the treatment of other diseases also, when it becomes available.

The drug has no toxicity, is well tolerated, has no bad taste and is effective in very small doses. We hope that the price also will be quite reasonable.

I have no laboratory facilities and the cases have been diagnosed purely on clinical symptoms, naked eye examination of the stool and the presence of a mild epidemic of dysentery in this part of the year in this locality.

I express my gratitude to the Superintendent, Bengal Immunity Co. Ltd. for supplying free samples of the drug for trial in all these cases in my clinic.

A CASE OF ANÆMIA TREATED ON INTRAVENOUS IRON THERAPY

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Name of the patient: A.D., aged 25 years; residence: Delhi. Patient started attending O. P. D. from 13-9-'50.

Complaint:—Increasing weakness for the past several weeks. Pregnant: 8 months. 5th para. Three were full term normal deliveries but none living. Two of these children died one on the 15th day and the other at 3 months. One was twins. None living.

On examination the patient was a pale looking woman. Teeth and tongue—N.A.D. P.A.—Uterine size 8 months L.O.A. F.H.S. good. Liver and spleen not palpable. Heart—both sounds faint. No adventitious sounds heard. Blood pressure —110 and 60. Hb. 50% by Hæmoglobin scale book. Her blood was ± for Kahn. Stool—showed no abnormality. Urine—N.A.D.

She was given Procaine Penicillin 4 lac units one dose and a course of six injections of Thiarsin. She was also put on Folvron by mouth. Injections Neohepatax 2 c.c., Vitamin B complex 2 c.c., on alternate days. These were continued throughout but hæmoglobin remained persistently below 50%.

Previous to starting treatment in the hospital she had taken a lot of treatment from private doctors but no improvement occurred. She was admitted on 2–11–'50, delivered a female child weighing 6 lbs. 12 oz. Her hæmoglobin was 30% by Hæmometer. The same two injections were continued but Inj. Iron Arsenic were also given. During her stay of a week in the hospital she was also put on large doses of Ferri et Ammonia Citras. She left the hospital against advice. She then started taking injections of Vitamin B_{12} but with no improvement whatsoever. She was advised blood transfusion. She consulted me again in December for increasing weakness with attacks of faintness and giddiness.

The blood examination report was as follows:—R. B. C. count: 2,410,000. Hb. % Sahli 30% 5·16 gms. per 100 c.c's. Colour index ·6. W.B.C. count: 9050; Diff. count: Polymorphs 72%; Lymphocytes 21%; Large Mononuclear 3%; and Eosinophils 4%.

Before taking blood transfusion I advised her to take intravenous iron injections. I started with 2 c.c's of Iviron undiluted but given very slowly. She felt faint for 15-20 minutes. She was made to lie down and given a few drops of Niacin.

She was next given 5 cc. and was continued on 5 cc. on alternate days, or even on 5th or 6th day, depending on the reaction. It was very difficult to give her an injection intravenously as the visible veins were threadlike. I persuaded her to take at least six injections, by which time she could decide for herself whether she should take more injections or not. After the fourth injection she felt definite improvement and her attacks of faintness disappeared. Incidentally her veins also became more visible and injections became easier. After six injections her Hb. increased by 10%. She was given 12 more injections of Uniferron 5 c.c containing 100 mg. of sachharated Oxide of Iron. Her Hb. went upto 60%. There was a little setback in between the injections due to a bout of malaria.

This patient had tried all kinds of known treatments for anæmia, *e. g.*, large doses of Ferri et Ammonia Citras, Vitamin B Complex, Liver, Folic Acid, Vitamin B_{12} etc., and showed no improvement with any kind of treatment but with intravenous iron therapy she started remarkable improvement.

This case is therefore quite illustrative. I have since then started giving Uniferron injections to every indoor anæmic patient and in every case there has been improvement. In some it is slow, in others rapid, but in none of the cases I have found Hæmoglobin going up by 4% after 100 mg. of Oxide of Iron as is mentioned in the literature and by the firms manufacturing these preparations.

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PLANNING FOR HEALTH

We have been stressing with all the emphasis we can command in the Antiseptic that if the schemes that have so far been formulated and are likely to be formulated in due course by the Planning Commission to bring about far-reaching improvements in the industrial, agricultural, economical and social spheres in the country, it should, side by side, give deep thought and consideration to the improvement of public health in the country. Only when the foundation is well and truly laid can the superstructure stand any stress and strain. So also in regard to a country, only when effective steps are taken to ensure positive health to the people, can they take an increasing and enlightened interest in the implementation of the plans for regeneration that have been formulated. We are glad that the Planning Commission has realised the importance of this matter. Addressing the meeting of the panel on health programmes held in New Delhi on April 12, 1951, Shri Gulzarilal Nanda, former Labour Minister of the Bombay Government and Vice-Chairman of the Planning Commission, recognised the importance of the health programmes of the future keeping pace with the growing expectations of the people under a new democracy in India. As the old order of alien rule in India has given place to a new democracy, it is essential, he said, to approach all social problems, including the problem of health, from a new angle so that concentration of efforts might be directed largely towards the benefit of the common man.

It is ironic that when our exchequers, Central and Provincial, were overflowing, our alien rulers had not the will to remedy the evils which deprived the common citizen of a fair chance of good health. Now we have the knowledge and the will too, but economic

and financial difficulties stand in the way. SHRI NANDA pointed out that while in the United Kingdom the percentage of expenditure on health to per capita income was as much as 3·9, it was as low as 0·6 in India. The percentage of expenditure on medical and health services in the Centre, States and local bodies in relation to the total expenditure comes only to 5·2 as compared to 10 in the United Kingdom. And the present is, no doubt, a great improvement on the past, due to the efforts of the Congress Ministries to provide for larger sums under this head. But the present provision is only a drop in the ocean. Yet the financial position of the Centre and of the States being what it is at the present moment, it is not possible for the Governments to provide larger and larger sums for the improvement of public health and the extension of medical relief far and wide. The Public Health Minister suggested to the panel that it may consider two methods for raising funds, namely by introducing health stamps as has been done in France, Belgium, Italy and Switzerland, and secondly by developing the idea of health insurance. As regards the first, India is not in the same position as other countries mentioned by her. The per capita annual income is much lower than that in those countries and the majority of the people are not even having one square meal a day. Besides, taxes have been raised considerably during recent years that it would be no exaggeration to say that people are really groaning under heavy taxation. It is therefore for the Planning Commission to consider if it would be wise to expect any large income from this indirect means of taxation. As regards the second, it is a matter worth the serious consideration of the authorities and the public.

In the circumstances the panel was called to discuss and advise the Planning Commission on the general pattern of health dvelopment, the priorities to be given to various schemes, adoption of a uniform health policy, co-ordination between the Centre and the States on health matters, and financing of health schemes and their re-orientation having regard to the special conditions and limited resources of the country. Shri NANDA placed before the panel several suggestions such as the vexed question of multiple systems, prevention of adulteration of drugs, improvement of health and vital statistics by application of scientific methods of registration, the chalking out of a programme for dissemination of knowledge on health and expansion of health education. He also referred to the present maladjustment in the sphere of medical education as a result of which "while, on the one hand, there are not enough doctors to go round the country, on the other hand, there is no lack of unemployment in the profession of medicine and nursing." He also made it clear that health services should be of real help to the poorer class and should be easily available to them.

The Health Minister availed herself of the opportunity afforded by this occasion to warn the panel against any idea of expanding

medical services at the cost of lowering the standards or quality of the profession. "The health of the nation is," she said, "its greatest asset and all systems of medicine must stand the searchlight of scientific investigation. Only those who are well equipped to practise should be recognised by the State." This was urged also by Dr. P. S. SRINIVASAN, at the Licentiates' Convention held at Ambala in April. "As matters stand now," he said, "there will be a clash between members of the scientific medical profession who number about 55,000 and the practitioners of the indigenous systems of medicine who, according to the Chopra Committee Report, numbered 2,00,000. It is essential to decide whether Government may safely let loose 2,00,000 practitioners of indigenous systems of medicine in India to do both preventive as well as curative work." The panel has divided itself into 11 sub-committees to consider problems connected with education and research, relative position of different systems of medicine, moral health, problems of malaria, tuberculosis and leprosy control, industrial health, woman and child welfare, health education, production and supply of drugs and other equipment, population growth and family planning. On the subject of family planning, the Health Minister suggested that the problem had to be tackled from a much more fundamental angle that what artificial methods of birth control could hope to achieve. Large scale introduction of artificial control measures, she said, apart from their doubtful moral value, was rather impracticable in India because the country could not just afford it. She herself made some suggestions such as the raising of the age of marriage, prohibition of marriages for persons suffering from insanity, leprosy and tuberculosis, provision for compulsory medical examination before marriage and finally steps towards improvement of the standard of living. The cumulative effect of all these measures, she hopes, would promote limitation of families. We shall await the reports of the Sub-Committees and the decision of the panel thereon with interest. The machinery has been set in motion and is bound to produce results.

THE LICENTIATES' CONVENTION

"TIME and opportunities are now ripe to do all we can to be forgotten as a separate entity. We should therefore not lose the time and opportunities at our command", said Dr. P. S. SRINIVASAN, M.L.C., Madras, presiding over the All-India Licentiaties' Convention held at Ambala in the second week of April 1951. If the profession as a whole has not become one entity so far, the fault lies on the All-India Medical Licentiates' Association, some of the leaders of which are still against the merger of the I.M.A. and the A.I.M.L.A., in spite of the fact that opinion is slowly and steadily gaining ground among the licentiates themselves that merger would

be to their advantage in every respect. The reasons are not far to seek. The Indian Medical Association has extended its hand of fellowship wholeheartedly; the Government of India themselves are veering round to the view that the differentiation which was created by their predecessors to suit their own purposes, would not pay, and that if the wholehearted co-operation of the profession is to be secured in the huge task of reconstruction that lies ahead of them in the country, *viz.*, taking effective steps to assure positive health to the people, the existing differences should be done away with; in fact, in this very Convention, the Hon'ble RAJKUMARI AMRIT KAUR, India's Health Minister, announced that "the question of instituting an All-India Register for doctors by amending the Indian Medical Council Act is receiving the urgent attention of the Government of India. Both graduates and licentiates would be eligible for registration"; the Government of Madras have removed the distinction between Civil Assistant Surgeons belonging to Classes I and II and placed them all in one cadre, though in regard to pay there is still a differentiation between the graduates and licentiates. Such a distinction is wholly uncalled for and unjustifiable in view of the fact that the licentiate course has been abolished in this State long ago during the first Congress regime and those of the licentiates who are now serving as Civil Assistant Surgeons are persons who have put in service for a number of years and have proved themselves as worthy as the graduates themselves. Further representation is bound to wipe out this difference also and if the demand goes forth from the profession as a whole the mistake is bound to be rectified early and licentiates in the cadre of the Civil Assistant Surgeons placed on a par with the graduates in regard to their emoluments. We therefore fail to see why the President, who is anxious that the licentiates should get merged in the profession as one whole entity, should still remind them that they are only a part of the whole and not the whole and that they should carry on their fight to get their grievances redressed. It is this lead which he gave, we think, that should have been responsible for the authority given to him by the Convention to set up an Eleven Man Committee in order to get their decisions implemented by the authorities. The royal road lies before them. By giving up their separate existence and merging the A.I.M.L.A. in the I.M.A. and making their cause the cause of the profession as a whole, they can get all their legitimate grievances redressed quickly. No Government, much less a responsible Government, can ignore the united demand of the profession.

Dr. SRINIVASAN urged the resumption of recruitment of licentiates for temporary military service through the I.A.M.C. This was one of the allurements offered to the licentiates by the previous Government during the war period to make the licentiates stick fast to their separate existence to suit their purpose, and this was dangled by those at the helm of the A.I.M.L.A. as one of their greatest achievements. We had our own doubts then if it would remain a

permanent gain, since no successor government was bound to follow it and we gave expression to it at the time. It has happened as we had expected and the recuritment of licentiates had been discontinued. We are at one with the licentiates in their demand, since they have, by their meritorious and gallant services during the emergency period, proved themselves fully worthy of the trust reposed in them, and we are sure the Indian Medical Association also will support them in this demand. The demand of the Convention that provision be made for the admission of licentiates in the State Medical Colleges for the condensed M.B., B.S. course is very just, reasonable and in the interests of the country itself. The more they learn the more their services would become invaluable. There is already provision for such a course in Madras and in Calcutta, and it is the duty of the Government to see to it that adequate provision is made in all the State Medical Colleges for this higher course for the licentiates.

Dr. SRINIVASAN had some harsh things to say against the present Indian Medical Council and its functioning. "Whatever might have been the practice during the period when India was under the yoke of foreign rule," he said, "it is humiliating that at present when India is independent we should allow the British Medical Council to continue directly or indirectly its authority on the Medical Council of India." Whatever might have been the inner motives of the previous Government to assign a dominating position to the British Medical Council, the reason openly stated then was the fixation of medical standards in India. Our country has achieved independence and it is as competent as the British Medical Council to fix its own standards in conformity with the standards prevalent in other countries. As a matter of fact, the Health Minister herself has been persistently insisting upon a higher standard, not to satisfy the Medical Council of Great Britain but to establish standards for ourselves in the best interests of the country. Now that the amendment of the Indian Medical Council Act, to enable licentiates to get themselves registered along with graduates in an All-India Register, is under the active consideration of the Government, it is worthwhile taking this matter also into consideration and freeing the Indian Medical Council from the domination of the British Medical Council and establishing our own standards.

The President appealed in fine to those who have retired from service in whatever capacity to start an All-India Medical Mission for service in the villages. As he said, 90% of the population of India live in villages. The state of affairs that existed during the British rule, the utter neglect of the health and welfare of the people in rural areas, still continued with this difference that while the British rulers, though they had the money, were unwilling to spend it for the improvement of rural areas, the present Government suffer under a serious financial strain due to causes into which it is

unnecessary now for us to enter. But this state of things should not be allowed to continue. The profession can do much to alleviate the sufferings of ailing humanity in rural areas. It is not only people who have retired from service but even those who remain in active service who can do much to alleviate their sufferings. And service to the poor, voiceless millions, is service to God. We would therefore suggest that the medical profession as a whole take a direct interest in the matter, evolve a plan of action and implement it. Such help as they may need for such implementation, the Governments are bound to give them and, we are sure, will give them.

Effects of Mass B.C.G. Vaccination

Measuring the effects of mass B.C.G. vaccination by the subsequent tuberculosis mortality involves long delay. This time lag, Dr. T. Gedde-Dahl suggests, might be overcome by studying the earliest manifestations of infection with tuberculosis. In Norway both erythema nodosum and pleurisy are notifiable: and since 1941 their incidence has declined remarkably as the following figures will show :—

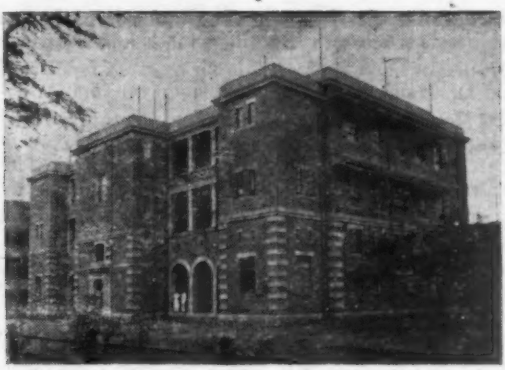
1941 ..	1374	2989
1942 ...	1416	2687
1943 ...	1155	2203
1944 ...	1192	2293
1945 ...	1073	2108
1946 ...	1147	1991
1947 ...	859	1708
1948 ...	634	1247

It will be seen that the fall in numbers was greatest in the last 2 years during which, for the first time, mass B.C.G. vaccination was carried out in Norway (35,000 in 1947 and 135,000 in 1948). There are, of course, various other factors for which credit can be claimed; during the past decade there have been large advances in the campaign against tuberculosis and since 1943 mass radiography has played an important part. The invidious task of apportioning marks between all the factors capable of reducing morbidity and mortality would tax the ingenuity of any statistician and Dr. Gedde-Dahl is not prepared to rush in where purists would not tread. *But he has a suggestion to make:* Let tuberculosis notifications be accompanied by a statement that the person concerned has or has not been vaccinated with B.C.G. In countries, where mass vaccination with B.C.G. is extensively practised, this plan might well yield valuable information.—(*Nord Med.*, 10-3-'50, p. 444, *The Lancet*, 29-4-'50, p. 838).

Diet in Congestive Heart Failure

Stroud and Stroud have suggested, on the basis of their experience in the management of congestive heart failure, that patients be put on a low sodium diet containing 0·5 to 1·5gm. of sodium per day. Low sodium diets are hard to follow because of the lack of palatability. Fluid intake should be adequate, atleast 2000 c.c. per day. The B complex and ascorbic acid should be given in adequate amounts. Protein 1 to 2 grammes per kg. of body weight is necessary. A caloric intake adequate for maintenance of body weight is also essential.—(*Med. Clin. N. Amer.*, 34, p 563, 1950).

HUNDRED YEARS OF PATHOLOGY*



(In the Grant Medical College, Bombay)

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It appears clear from all available records that the teaching of morbid anatomy took prominence very early in the history of the Grant Medical College. Records from 1872 of post-mortems done by several teachers have been carefully preserved and these will show the high standard of description attained then. It is, however, not possible to describe in so many words as to how the subject of Pathology was taught in the early years. In all probability, the work in the dead house formed all-in-all of Pathology, at least till 1877. In that year, on the 22nd of August, a memorandum was addressed to the Director of Public Instruction proposing a separate Chair of Morbid Anatomy and Pathology. In the same year a separate laboratory was inaugurated. The following extract from the College Annual Report will speak for itself :

"Work was commenced in the separate Pathological Laboratory. It is provided with all necessary apparatus, microscopes, preservatives, chemicals, re-agents and a series of registers in which the daily observations made in the dead house are recorded. This work is presided over by Dr. Anna Moreshwar Kunte, the Demonstrator in Anatomy. Several students, chiefly from the huge class of fourth year, have volunteered to do the daily routine work. Sir Cowasji Readymoney offered a prize to a student devoting to research in Morbid Anatomy."

Surgeon Arnott is mentioned as the first Professor of Pathology and Dr. Anna Moreshwar Kunte, the first Mayo Demonstrator in Anatomy. He was succeeded by Dr. Vithal Vinayak Gokhale in 1881 and Dr. S. B. Bharucha took up the post in 1897.

Government Resolution No. 1967, dated 4th June 1881, instituted a full-time Professorship of Pathology and Major Waters was appointed to that post.

To divert for a while, it was in the period 1890 to 1900 that several renowned research workers were given accommodation and other facilities within the four walls of this College. They all brought honour to the Grant Medical College.

Dr. K. N. Bahadurji (1889 to 1892) worked on the staff, in this period. A short life sketch of the pioneer worker by Dr. P. A. Dalal appeared recently in the Jan. 1949 issue of the *Indian Journal of Medical Sciences*. In 1895, Dr. N. F. Surveyor was allotted accommodation in the Framjee Dinshaw Petit Laboratory

* Some of this account has appeared in the Grant Medical College, Magazine, Vol. xlix, pp. 4–12, Dec. 1949.

for Medical Research. Dr. Surveyor was the Professor of Bacteriology for many years. His notable researches were on actinomycosis and malaria.

In 1896–1897, arrangements were made for research facilities for Messrs. Haffkine, Hankin, and Dr. Manser—well-known plague workers.

In 1898–1899 Messrs Marsch and Pitchford were provided for in the Petit Laboratory for plague research. In the same year, Dr. R. Row was allotted a set of rooms for research in the same laboratory. Dr. Row worked in these rooms till 1929. Dr. Row's record of research papers has rarely been surpassed.

The rooms used by the late Dr. Surveyor were, after his death, used for short periods for research by Dr. A. J. Noronha and Dr. L. D. Dhawale.

It may be mentioned here that the post of Bacteriologist, for the first time held by Dr. Surveyor, came into existence in 1906–'07. Dr. D. A. Turkhud was appointed Professor of Bacteriology in 1914-'15, and Dr. P. A. Dalal in 1924. The Professorship was subsequently abolished, being replaced by a lecturership and that, as an independent post, at a later stage, was also abolished.

Thus at present there is a single post of Professor of Pathology with lecturers under him.

The Pathology Laboratory work was carried out for a number of years in a part of the ground floor of the college main building. Post-mortems were conducted in a room adjoining the wing where the present Pharmacy Department is located. The post-mortem work was later shifted to the present ladies' waiting room, from there to a shed behind ward No. XIII, and thence to another place, a stone building, now used as stores, and finally in 1909 to the present post-mortem room. This is about the best connected account that can be collected. While post-mortem examinations were being carried out in the present ladies' waiting room nearer the road, Koch worked and demonstrated the cholera vibrio. It was again here that Childe described the lung of pneumonic plague.

With the completion of the Coroner's Court building in 1909, the Pathology Department "secured accommodation better than it ever had before". The first floor nearly 4000 square feet, was used by the Pathology Department and the ground floor was occupied by the Coroner and the Police Surgeon much in the same way as one sees now. The present practical class room on the first floor has remained practically unchanged. The present lecture theatre is a new design remodelled in 1929. This room alone was all that formed the Pathology Department from 1909 to 1929. This large room was the working place for the staff and technical assistants, of whom there was only one. The Professor had a room to himself, the present preparation room.

The staff was not very elaborate, – a Professor, a part-time Assistant Professor and two part-time Tutors. The Bacteriology Laboratories were housed independently in the present students common room. The total number of laboratory examinations was comparatively small, never exceeded 500 to 700 in a year. There were many autopsies. All permanent records were written up by students, till 1932. All records from 1872 are available and all records have been indexed. The course of studies was simple, the tuition proportionate, and the degree examination consistent with all these. There was no separate higher degree in Pathology and Bacteriology till 1928. Mention must be made at this stage that the Sir Jamshedji Jijibhoy Hospital had only two laboratories (of the status of clinical side-rooms) quite until 1934, one on the ground floor and the other on the 1st floor. These were used by the medical officers and by their registrars (the present housemen) as they were then called and by the students. It was in one of these laboratories, the one on the first floor (the Laboratory No. 1 of to-day) that the great discovery of the relapsing fever spirochæte was done by Dr. Vandyke Carter, without the help of an oil immersion lens (1877–1880). Carter's book, Spirillum Fever, is a famous publication. He worked on mycetoma, rat-bite fever, urinary calculi and several other subjects. Carter donated Rs. 10,000 and founded the Lord Reay lecturership for encouraging research in Physiology, Pharmocology, and

Pathology. 1914–1918 saw the World War I. This period interrupted the growth of this scientific subject in this college. Several officers were called up, many of the graduates joined active service, and so great was the shortage of medical men that students before getting a degree were selected to occupy posts of registrars, posts parallel to the present day housemen.

Mental strain coupled with scarcity of men and materials produced a deleterious effect. Progress of the work stopped and at the end of the War, as far as I can recollect, the Pathology Department was behind where it had been in 1909 and much farther behind what the world expected it to be then. The five years after the War were years of new schemes. The re-staffing of this institution took birth.

During the four years of the War and the few years after it, a series of changes took place in the staff of the Pathology Department. Major Gorden Tucker, Major L.T.R. Hutchinson, Dr. F. N. Kapadia and Major S. S. Vazifdar worked as Professors.

It was at this stage that Dr. V. R. Khanolkar, M.D. (Lond.), B.Sc. (Lond.), was selected for the post. He took up the post and started improvements. He made proposals for the expansion of the buildings, etc. About this time the scheme for the opening of the Seth Goverdhandas-Sunderdas Medical College, Parel, Bombay, matured and Dr. Khanolkar decided to leave the G. M. C. to join the new college which was to have an entirely Indian staff. The new college paid higher salaries too. Before he had completed two years of service in the Grant Medical College, Dr. Khanolkar left.

The author of this article had joined the Pathology Department as a voluntary worker in 1925, has been appointed Assistant Professor later and had the honour to succeed Dr. Khanolkar as acting Professor in 1926. It has not been an unbroken period of responsibility on my shoulders since then. Almost a year later, Government requested Dr. Row to accept the Professorship. Dr. Row retired in 1932 and I succeeded him and I am holding my present post since then, actually from the 16th of December 1931. Thus I have nearly completed 25 years connection with the Pathology Department as a member of its teaching staff. Long connection with a Department brings with it responsibility. Whether I have fulfilled a useful purpose future generations will say.

I propose to give below an account of the growth of the Department of Pathology during this period.

With the opening of the G.S.M. College, the Grant Medical College, in a way, lost the monopoly of creating medical graduates. The new college had the advantage of all the mistakes the G.M.C. had committed and consequently made a good start.

The Pathology Department, as it was housed in a small place, found it impossible to cope with the ever-growing ambitions of the reorganised staff of the J. J. and allied hospitals. Schemes for re-staffing were put up very early in 1928. The anticipated expense was great and therefore Government sanction was difficult to obtain even in those days. When a case was made out it was referred back for convincing arguments and yet more convincing arguments and the time spent in writing up these, easily enable one to bridge over financial years. 1943 saw some of the proposals first put up in 1928 fulfilled, and even up to the date of writing this staffing is under discussion. Proposals for additional accommodation were under consideration for years. Twice proposals for a large building for Bacteriology were made. Tentative plans for additions to and alterations in the existing Pathology building had also been made.

It was not until Dr. Row's personal request to Sir Dorab Tata that a further definite step was taken. An offer of 2 lacs was made to the Government by him. An equal amount was to be contributed by the Government.

A committee of officials and non-officials was appointed by the Legislative Council to examine the plans. These were finally approved. The building was completed in less than 18 months and was formally opened by the Surgeon-General, General Mackie on 7th June 1929.

This gave the Pathology and Bacteriology Departments of the College a House. The two Departments were moved into this Pathology School under one roof. The new building housed well the Museum which till then occupied a part of the first floor of the college main building. For the better unification of efforts it was considered necessary to introduce a certain degree of amalgamation of the two Departments. This was effected soon after occupying. The abolition of the Professorship of Bacteriology which came much later, it can be assured, will not hinder the growth of the subject of Bacteriology. In fact, with proper adaptation and co-ordination, the growth ought to be, and I assure will be, faster and healthier.

From the moment of occupying the new building, from time to time alterations in the use of different parts of the building has been found necessary. Changes of a definite nature towards improvement in the staffing have been effected in spite of the War, and the Department was distinctly better off than it was when I left it in 1941 for active service. There are further schemes for improvement under consideration. Additional staff has been administratively approved in 1950. The teaching department has not grown lobside. The clinical investigation section has materially been reorganised. In 1934, a wholesale reorganisation of the ward laboratories was done, providing a clinical side-room per 30 beds. This proportion was unfortunately upset during the War, mainly due to shortage of space and materials. It has been re-established in recent years. Thus in place of 2 laboratories there are now functioning numerous clinical side-rooms for the use of housemen and students.

The Vandyke Carter Central Clinical Laboratory established in 1933 has been replanned to function better as new staff is arriving. It is the ambition to make this laboratory a training centre for students.

The designation of the Head of the Department of Pathology up to 1934 was Professor of Pathology, Morbid Anatomy and Histology and Curator of the Museum, Grant Medical College, quite a descriptive phrase.

This alone can floodlit the growth of the Department. At one time there was a Curator of the Museum and a Professor of Morbid Anatomy. Step by step, duties of several were combined and produced this clumsy looking designation.

It was at my suggestion that two changes were effected by Government in 1934. The designation was changed to Professor of Pathology and Pathologist, J. J. Group of Hospitals. The second change was making the post a non-vacation one ; and today the rest of the full-time posts are also non-vacation posts.

For a period of almost 30 years the Department had a part-time Assistant Professor. His salary was Rs. 200/- per month. For the first time in 1938 the post of the Assistant Professor was made full-time. The post was included in the B.M S. Class II. At the beginning it was non-pensionable and temporary. Much later it was made pensionable as it is now. The designation and scales of pay are now revised. The present pay scales, it is hoped, will attract keen persons.

There were two tutors in this department till 1938. They were paid Rs. 100/- and Rs. 75/- per month, depending on their being senior or junior. These posts were attractive at one time, many first class graduates took these up. A certain time limit for the tenure was introduced after 1926. Till then a tutor could hold an appointment as long as he wished. In 1938 these posts were replaced by those of part-time demonstrators.

Three demonstrators were appointed on Rs. 50/- per month in place of the two tutors. At the time of writing two classes of demonstrators exist (i) full-time and (ii) part-time. The Department was given three assistants satisfying the demands of the University. It may be said that the chief reason for the changes in the staffing has been the need for adequate provision for teaching. The College possessed a Museum from the early days. The following are some milestones in the growth of the Pathology Museum of this College.

The first Museum Catalogue was published in 1850. The Museum had a full-time Curator for 30 years upto 1877. At present the Museum occupies a floor space of 11,200 sq. ft. There are several sections—general pathology, special pathology, hygiene and preventive medicine, forensic medicine, etc. Needless to say, building up of a Museum needs help from all quarters. The College Museum is referred to by Markham and Hargreaves in the volume "Museums in India" published by the Museums' Association, London, 1936. Now I turn to the present.

The Department, as it is now, the Department of Pathology and Bacteriology, has a single head controlling the clinical investigation work for the J. J. Group of Hospitals and the teaching at the Grant Medical College, in collaboration with the clinical staff of the J. J. Group of Hospitals. The clinical investigation department has two full-time resident clinical pathologists and some technical staff. The work is carried out in three stages; bed-side investigations are done by housemen, further work done by the Vandyke Carter Central Clinical Laboratory in the out-patients' department building, and work beyond the capacity of this unit is passed on to the Pathology School. In this building on the first floor a set of six rooms is set aside for clinical investigations. In these rooms are being carried out bacteriological, hæmatological, chemical and histological investigations.

The work at present is heavy and is expected to grow still more.

Teaching of undergraduates.—There is adequate accommodation for practical classes and lectures and lecture-demonstrations. There is the necessary equipment and teaching material. Post-graduate teaching efforts are being made to satisfy the ambitions of post-graduates. Much more requires to be done for adequate facilities for post-graduate study in this as in many other subjects.

The Department possesses an animal house and necessary animals are available.

Adjoining the Pathology School is the mortuary with a cold preservation plant. This was built along with the Pathology School in 1929. In 1940 a shed for mourners was built.

The six years of the World War II has fortunately left institutions in this country intact. With the exception of the damage done by the explosion of 1944, the buildings of the Pathology Department have remained in good condition. The work was carried out by the staff between 1941 and 1946 under Drs. Paramanand, Manohar, Saldanha, and Parekh as the head. These years were of continued strain and difficulties all round and all praise to these men who continued to do good work under such conditions.

Research work in the Pathology Department and research work by the staff have been subjects of much criticism in recent years. Plenty of clinical material is available and there is adequate accommodation. Equipment, as and when required, is obtainable. Still one cannot deny the fact that these latter 20 years have not been as productive as the early period wherein persons like Vandyke Carter, Dimmock, Hatch, Haffkine, Row, Surveyor, Childe and Choksy, worked and produced work recognised even beyond the length and breadth of this country.

Belonging to the present generation, I must plead guilty to the charge.

Analysing the causes may be of some use to the readers. Men who worked have been capable. Endeavour, skill, desire to work and labour have not been lacking. Circumstances have been unfavourable. The nature of present day medical research is totally different.

The staff of the Pathology Department in outline remained unchanged for over 50 years though extensive extensions took place in the hospitals, the Department was responsible to serve. The University Regulations became more exacting and so also did the modern world. The demands, increasing without parallel additions to the personnel and finances, placed the Department in an almost impossible situation. These latter 20 years have been of growing ambitions and of unprecedented strain on the few men who strenuously worked to make the two ends meet.

Routine work is despised by some who think of research in terms of the fundamental sciences. Medical work involves many applied sciences and modern medical research therefore has to be a team work. Very high standard of routine work must be the basis for any institution to aspire to produce research. Our efforts in the last quarter of a century have been devoted to the many problems of maintaining this high standard. This will provide the background for further work.

A Society of Pathologists was started first in India in Bombay, thanks to Dr. Khanolkar for his lead and continued co-operation. Regular meetings have been held for over 14 years. The standard of papers read by the staff of the local institutions and post-graduates is undoubtedly such as to make one feel proud of. Nearly 100 meetings have been held so far, about half of which have been at the G.M.C. Papers published by this Department have been reviewed by the press, and quoted by standard authors. Drs. Row, Parmanand, Saldanha, Parekh, Manohar, V. V. Gharpure, Soman, Deshmukh, Tilak, Jhala, Salaskar, Mrs. Soman and others have contributed several papers of scientific interest in the Medical Journals. The total number of papers published and read at scientific meetings exceed one hundred. This has been in the period from 1926 to 1945, a combined effort of all of us of the Pathology Department. Yet one feels one ought to have done better with all the wealth of material available.

Let us then be proud of our heritage, let us be constructive in our criticism determined in our daily effort now, to lay the foundation for a more hopeful and prosperous future for the Motherland !

Gleanings From Medical Press

SURGERY

Six axioms to remember in acute abdominal emergency.—(*Amer. Jour. Surgery*, July 1950, p. 96).

1. Severe abdominal pain of six hours' duration in a previously well individual usually indicates surgical intervention.

2. If any patient with abdominal pain is found to have a temperature of 104 to 105 F. at the on-set of illness the thorax or kidney is very likely the seat of the disease.

3. A normal pulse does not necessarily indicate a normal condition of the abdomen, especially in the early stages of an abdominal emergency.

4. Irritation of the pelvic nerves causes no abdominal wall rigidity.

5. Movement and pressure increase pain in peritonitis but afford some measure of relief in colic.

6. The stethoscope is as important in surgery for abdominal auscultation as it is in internal medicine for auscultation of the chest.

Treatment of infections of feet in diabetics.—(Butler. E. C. B., *Lond. Hosp. Gaz.*, 53, 3, 1950).

In the clinical supplement, to the London Hospital Gazette, Butler, has recorded his observations on a series of cases admitted into his wards at the London Hospital, during 12 years ending 1949. He divides his cases into 2 groups :—(1) Patients who have all signs and symptoms of the socalled senile obliterative arterial disease, and in addition have infection as a constant feature.

(2) Cases of diabetes complicated by infected feet ; usually circulation is good and the popliteal artery can readily be felt. Oscillometer tests show good pulsation below the knee.

GROUP I.—Following irritation or mild trauma a small infected area starts on one or more toes, also sometimes on heel or elsewhere on the foot. The infection may resolve on treatment or may spread or proceed to an area of necrosis which in turn may be localized

or spread to the whole foot. Pus formation is unusual and no definite line of demarcation between healthy and diseased tissue is visible. These patients have signs of thrombosis in the femoral or popliteal vessels. Radiography often reveals widespread medial calcification of the main vessels. The average of the patients in this group was 64.

Treatment:—Penicillin-resistant organisms may be present. So a combination of drugs has often to be chosen. Affected limb should be rested on a soft pillow at room temperature; both heels should be taken care of to prevent pressure necrosis. Active physiotherapy to both limbs should be given to aid circulation. Slough should be removed and necrotic phalanx cut away. Formal amputation of toes through metatarsophalangeal joints is contra-indicated as it leads to rapid spread of infection and to necrosis. Local application of penicillin-sulphonamide powder is of no use as it cakes and prevents penetration. Hypertonic saline dressings or Eusol compresses are useful; part should be kept slough-free and dry; a spirit dressing is very good. A course of systematic penicillin is valuable as it prevents cellulitis; the infection will subside in most cases and necrosis may be limited to one toe; if patient is fortunate the dead area may separate spontaneously. The indications for amputation in this group are spreading necrosis, intolerable pain and fulminating infection. 35 out of 40 patients in this group recovered under this regimen.

GROUP II—CASES WITH A GOOD POPLITEAL PULSE:—As blood supply in these cases is good, infection rarely leads to gangrene except where gas infection supervenes. Abscess formation is common and infection readily tracks up the layers of the foot causing sloughing of the tendon and fascia. Osteomyelitis with sequestration of affected toes frequently occurs.

Many of this group also have diabetic neuritis, as shown by perforating ulcers commonly at the ball of the big toe. X-ray shows underlying infective arthritis and osteomyelitis of the adjacent bones. The ulcers may remain quiescent for a long time except for a painless chronic discharge. A few cases in this group suffer from increasing blindness due to diabetic retinopathy. Every effort must be made to avoid a major amputation on these patients, as it is difficult for them to use crutches. Butler found that retinopathy and diabetic neuritis occurred only in group 2 cases.

Treatment (of Gr. 2):—Chemotherapy is likely to be more useful than in Group 1, but an accurate bacteriological diagnosis should be made. Systemic penicillin with a course of sulphonamides has yielded good results. Aureomycin will be a most valuable addition in the most severe types of anærobic infection. Abscesses should be drained freely and incisions must be adequate. The webs between toes need draining and the incisions must be prolonged on to the dorsum or sides of the foot. Drainage *via* the sole should be avoided as far as possible. Formal amputation of the diseased toes or metatarsals can safely be performed once the acute phase is over. These wounds may be sewn up with confidence, after all dead tissue has been removed. Systemic pencillin should be given in these cases till the wounds have healed.

The principles of treatment adopted in this series of 61 patients (with only 9 deaths) were:—

(1) Infection to be controlled with large doses of penicillin and if resistant with aureomycin.

(2) Immediate surgical intervention is unwise and as soon as pus has formed the drainage must be carried out.

(3) Emergency amputations before the infection has been controlled are dangerous and may be fatal.

(4) Should amputation be required the flaps must *never* be sutured; they should be loosely tied over a pack; secondary suture can be done on the 5th day if infection has not spread.

(5) Blood transfusions are valuable to counteract the progressive anæmia, usually present.

It was impossible to control the glycosuria in these patients until the infection had been overcome; the demand for insulin then fell with dramatic suddenness.

Carbohydrate required to prevent post-operative ketonuria.—(*Surg. Gynaecol. Obst.*, 90, p. 349, 1950).

The urines of 68 post-operative patients on varying parenteral carbohydrates were tested for the presence of acetone and glucose during the first three post-operative days. After operation under *spinal anaesthesia* the majority of patients receiving 100 or 150 gm. glucose a day as the sole source of nutrient showed acetonuria while the majority of those receiving 200 to 300 gm. glucose daily did *not* show acetone in the urine. After operations under *ether*, the incidence of ketonuria was much greater at a daily carbohydrate intake than it was after operation under spinal anæsthesia at the same intake. *This indicates the superiority of the spinal anaesthetic for diabetic* patients and those with liver disease in whom the maintenance of liver glycogen and the avoidance of ketosis may be a problem. An average of 4 to 6% of the glucose given intravenously was excreted in the urine, but there were marked and unpredictable variations in glycosuria from patient to patient.

Conservative treatment of massive gastro-duodenal hæmorrhage.—(*Am. J. Surgery*, 80, 7, pp. 883—887, Dec. 1950).

The remarkable hæmostatic effect reported by the use of gelfoam and thrombin in neurosurgery and urologic surgery prompted Cantor and his co-workers of the Detroit General Hospital to investigate the value of these two hæmostatics in gastro-duodenal hæmorrhage. After successful animal experiments, they have used these in the treatment of 73 consecutive cases of massive gastro-duodenal hæmorrhage. All patients admitted were immediately seen by the resident surgeon and the following routine was promptly instituted. (1) History is taken and physical examination made to ascertain ætiologic basis for the gastric hæmorrhage. (2) Complete blood count is taken. (Dyscrasias are thus ruled out by tests). (3) Shock, if present, is next treated. (4) Blood pressure and pulse readings are taken every 2 hours for the first 48 hours. (5) Blood is replaced depending on the patient's condition and RBC count. Patient in shock is given 1000 c.c. of whole blood at once; patients with RBC count below 2 millions are given 1000 cc. and those with over 2·5 millions get 500 c.c. Blood transfusion is made as often as necessary; as much as 10 litres may be given in 24 hours. (6) In addition to adequate blood replacement and the conservative routine of Meulengracht's diet, antacid therapy, sedation and bed rest, the only new and additional therapy is the use of gelfoam and thrombin. (7) *Gelfoam is prepared as a fine dry very light powder and 2 tablespoonfuls of gelfoam are mixed with 2 ounces of milk and cream and given to the patient orally *every 2 hours*; immediately following this, 250 units of thrombin (50 cc. of a solution containing 1000 units of thrombin dissolved in 200 cc. of water) is given after the gelfoam. (8) Amphojel (Aluminium hydroxide suspension) is given (following thrombin solution) in a dose of one tablespoonful every 3 hours.

There were no deaths in this non-surgically treated group of 73 patients. Gelfoam and thrombin are therefore very useful additions to our armamentarium in such cases.

*(Gelfoam supplied by Upjohn Company, Kalamazoo).

OBSTETRICS AND GYNÆCOLOGY

Streptomycin therapy during pregnancy.—(*Proc. National Acad. Med.*, Paris, 1950).

Bernard, Kreis and their co-workers reported to the National Academy of Medicine certain very important observations on the relative values of streptomycin and dihydrostreptomycin, in the treatment of tuberculosis in pregnant women. The tolerance to streptomycin was good, the daily dose was 1 gm. in 4 cases, 1·5 gm. in 8 and 2 gm. in 2. Total dose varied from 20 to 180 gm. With the collaboration of Mrs. Grumbush of the Pasteur Institute, they studied the transplacental

passage of the anti-biotic in 12 women who were given in the course of labour 0·5 gm. dihydrostreptomycin. The examination of the blood of the umbilical cord taken immediately after delivery, revealed the constant presence of the anti-biotic. The average rate observed between 43 and 130 minutes after the injection amounted to 5·7 units, results agreeing with those of other workers. Bernard and his associates believe that this placental barrier indicates the harmlessness of streptomycin for the fœtus. Prof. Moulonguet has some reservations regarding the children's future and recalled the recent report before the Society of Otorhinolaryngology, Paris, of a case of deafness following treatment of a child in this manner. He considers that on account of the difficulty of making an acoumetric examination in young infants, one *cannot* immediately state definitely that streptomycin therapy during pregnancy is harmless. For this reason, Bernard proposes avoiding administration of dihydrostreptomycin to pregnant women, reserving the streptomycin treatment for conditions with precise indications.

Placental transmission of chloromycetin.—(*J.A.M.A.*, 142, 17, p. 1361, 1950).

This is an important communication from the Research Foundation of the Children's Hospital, Washington. Ross *et al* carried out an experiment to determine whether chloromycetin passes through the placental barrier, like penicillin and streptomycin. An oral dose of 1 gm. was given to 8 patients and 2 gm. to 3 patients in active labour about one to 5 hours before delivery. Blood for chloramphenicol assays was obtained from the mother and cord at the time of delivery. In one case (out of eleven) twins were born and both were assayed. In 6 out of eleven patients, (including the twin case) chloromphenicol passed through the placental barrier in concentrations of 30 to 80% of that noted in the mother's blood. In the remaining 5, it was not detected in the cord blood; and in these the concentration in the maternal blood was 3·8 micrograms only per c.c. or less, while in the other 6 it ranged from 10 to 30. To ensure the diffusion of the drug into the fœtal circulation, it would appear necessary to maintain an adequate blood level in the mother of at least 10 micrograms per c.c. This could be achieved if a dose of 50 mg. per kg. of bodyweight per 24 hours in a four hourly divided dosage is given.

The significance of these observations assumes importance when chloromycetin is used in the treatment of susceptible maternal infections, such as, bacterial pneumonias, septicæmia, typhoid, ricketsias and genito-urinary tract infections. This anti-biotic has recently been acclaimed as effective in syphilis and hence would be effective in congenital syphilis and ante-partum when used instead of penicillin.

[Whether this drug will induce fœtal anomalies (such as occur when certain virus infections like maternal rubella are treated with this drug is not however known.] The empirical use of chloromycetin is however desirable, in the opinion of Ross and his co-workers, in order to avoid fœtal damage, in cases of atypical virus pneumonias.

Vitamin A in pre-menstrual tension.—(*J.A.M.A.*, 142, 16, p. 1312, 1950).

Argonz and Abinzano of the endocrinological service of the Revadavia Hospital, Buenos Aires, obtained successful results in 100 cases of pre-menstrual tension treated with vitamin A. Complete relief of mastodynia was effected. Abdominal pains decreased. Oedema, lumbar pains and nervous symptoms were not observed. The improvement of mammary disturbances was remarkable. A daily dose of 2,00,000 units is given orally during the second half of the cycle. The medication must be repeated for 4 or 5 consecutive monthly periods in order to obtain a prolonged result. Smaller doses and shorter periods do not give satisfactory results.

Prophylactic penicillin in obstetrics.—(*Am. Jour. Surg.*, 81, 2, pp. 168-171, Feb. 1951).

Dorgan *et al* of the department of Obstetrics and Gynæcology of the Naval

Hospital, Postsmouth (Va)., report on a study to determine whether or not the routine administration of penicillin to all patients early in the course of labour was an indicated procedure, calculated to prevent or minimise the incidence of puerperal infection and whether any untoward effects might be anticipated from such a routine. A total of 1131 vaginal deliveries was conducted from 8-3-1949 to 11-9-1949 and at the onset of active progressive labour, *i.e.*, 3 to 4 c.m. of cervical dilatation, 300,000 units of procaine penicillin G in sesame oil was administered intramuscularly to 509 of these 1131 patients. No one in this series received more than the one prophylactic penicillin injection. The alternate case method was employed and the only conscious selection was in placing in the control group those patients admitted to the delivery floor in advanced labour; this naturally placed a larger number of primiparas in treated group and multiparas in the control, but the difference was not significant. 622 did not receive penicillin and constituted the control group. 509 received penicillin prophylactically and comprise the treated group.

From the results of careful observations, it was evident that the prophylactic injection of 3,00,000 units of penicillin G in sesame oil early in labour resulted in a significant decrease in the incidence of puerperal sepsis and a considerable decrease in the number of febrile days. Morbidity and febrility were reduced by 70 per cent.

Systemic reactions to the anti-biotic did not occur in the series and local reactions were negligible; whether any patient in the treated series was sensitized by the penicillin, and whether any case of syphilis was there by masked and if any infant will suffer from its intrauterine dose of penicillin, are questions, though of not any serious or major import, are receiving due consideration from these authors and will form the subject of a later communication.

Hydriodic acid to overcome tubal occlusion in sterility.—(*Amer. Jour. Surgery*, 80, 1, pp. 131–132, July 1950).

In the treatment of sterility in women many cases of tubal occlusion are found to be due to the presence of thick mucus in the lumen of the tubes and to adhesions and scarring as the end result of inflammation. When repeated uterotubal insufflation fails to show patency of the fallopian tubes the outlook for the sterile patient is very dismal, or even hopeless. Yet women so afflicted continue to search for help. In considering what drugs might be of value in these conditions, Dr. Sharman S. Garrett of Illinois, hit upon the iodides as offering some hope for success. Iodides have the property of increasing the amount of secretion of the mucous membranes and of making the mucous secretion more watery and less tenacious. Iodides are used against thick sputum. Iodides are also believed to soften scar tissue—a resolvent action. Since these two actions are exactly the ones that might be of help in overcoming tubal occlusion, *Garrett* tried the use of a simple iodine preparation syrup of hydriodic acid on three patients who had persistent tubal occlusion, with success in two cases.

The patients were given syrup of hydriodic acid (two drachms) in water. In one case it was *given twice a day* after meals for two months and in another it was given four times a day for ten weeks. In both cases, tubal insufflation was done after three months, and was successful. The patients experienced shoulder pain; and the gas was flowing in good volume 30 to 40 mm. Hg. The first of these three patients became pregnant five months later. In a third patient aged 32, the iodide treatment for 2 months was not successful. She is undergoing further treatment. The two successful cases (aged 37 and 35 respectively) do not offer definite proof of the efficacy of a therapeutic measure; but it would seem worthwhile to give iodide therapy a trial in patients with persistent tubal occlusion. A warning is also given by the author:—"Iodide should not be used in any case suspected of tuberculosis because of the danger of its increasing the activity of the disease."

Further study of the effect of iodides

on patients with persistent tubal occlusion is warranted.

Chloromycetin in the therapy of granuloma inguinale.—(Robert B. Greenblatt, Virgene S. Wammock, Robert B. Dinest and Robert M. West, Augusta, Ga., *Am. J. Obst. and Gynec.*, 59 : 1129–33, May 1950).

Twenty-three women with granuloma inguinale were effectively treated with chloromycetin. The total dosages necessary for cure varied from 20 to 70 Gm. It appeared that the ideal dosage was 500 mg. every six hours for 10 to 20 days. If healing was incomplete or the lesions were extensive it seemed advisable to continue chloromycetin until 50 to 70 Gm. were given. Recurrences were few and these responded to a second course of therapy. Donovan bodies, in most instances, disappeared from the lesions in 2 to 4 days after therapy was begun. Untoward effects were not observed.—*Obstetrics and Gynaecology.*

Penicillin in the treatment of uncomplicated gonorrhœa.—(A. J. King, F. R. Curtis and C. S. Nicol, London Hospital, London, England. *Lancet*, 1 : 701–03, April 15, 1950).

It is pointed out in this paper that widespread optimism exists concerning the efficacy of penicillin in the treatment of uncomplicated gonorrhœa. The authors agree that penicillin is the treatment of choice in this condition but they stress the fact that cure is difficult to determine with certainty and that criteria vary in different clinics and may be quite unreliable through brief duration of observation and lack of detailed clinical and laboratory examinations. In male patients it is most important to examine an early morning smear and the urine which has been retained in the bladder overnight. This method often demonstrates evidence of residual infection. When obvious clinical relapse occurs with gonococci reappearing in the urethral smear or culture, then treatment failure is often attributed to re-infection, whether or not the patient admits to further sexual intercourse. In truth there is no way of deciding between re-infection and relapse, and it is safer in these cases to assume that treatment has failed to cure in assessing the value of a comparatively new drug. It is therefore suggested that all cases of gonorrhœa in which gonococci are found again within three months of treatment should be regarded as relapses.

There is little evidence to support the theory that penicillin treatment failure is due to 'penicillin-resistant' organisms; tests in vitro alone cannot be relied on for they cannot take into account the factor of tissue immunity in the host.

An analysis of 1788 male and 481 female cases is presented. All the females and 805 of the males had a first attack of acute uncomplicated gonorrhœa. All were treated with sodium penicillin (aqueous), 150,000 units given intramuscularly in five equal doses two-hourly ; 1447 of the males and 432 of the females attended for tests of cure. Ninety-four men (6·4 per cent) and 15 women (3·5 per cent) were regarded as immediate failures of treatment because gonococci persisted in the secretions or reappeared within two weeks, or because acute complications of the infection developed. In the remaining men, 276 (19·1 per cent), acute purulent urethritis developed during observation; 216 of them (14·9 per cent) showed gonococci in the secretions. A further 270 (18·6 per cent) showed evidence of residual infection in the genital tract either by rectal examination or by smear and culture of prostatic secretions. The total "late" failures in the male were 546 (37·7 per cent) ; if patients giving a history of previous gonorrhœa were excluded the total was 405 (27·9 per cent). Of the remaining women, 56 (13·4 per cent) showed gonococci in the secretions, while only 79 of 265 passed final tests of cure. In all the other cases there were abnormal clinical or microscopic signs in the urethral, cervical or rectal tests. The authors express the opinion that "re-infection" did not account for the major proportion of the failures. If all such cases occurring within three months of treatment are considered to be treatment failures then penicillin is less effective in the treatment of acute uncomplicated gonorrhœa than is generally accepted.

MEDICINE AND THERAPEUTICS

Typhoid and chloromycetin—Study of minimum dosage.—(Medina, I., Payne, E. H., Sharp, E. A., *Jour. Trop. Med.*, 53, 6, 199–125, 1950).

For communities in poor economic circumstances the expense of treatment is a considerable factor and, therefore, consideration of the most economical dosage that may be employed is important in order that the drug may become available to the greatest number of patients possible. Medina and his co-workers working in the Isolation Hospital at Guayaquil, Ecuador, treated 79 patients between March and Nov., 1949. Chloromycetin was not used for mild cases. Of 65 cases treated with one course of chloromycetin, one died and 64 were successfully controlled. Ages varied from 3 to 65 years. Of 11 patients who relapsed, ten were given either the same or a smaller dose than that used in the first course and they all responded promptly. 3 patients, on account of the seriousness of their condition, were given chloromycetin intravenously. It was dissolved in propylene glycol in a 15 per cent concentration and on an average 0·3 gramme was given every 3 to 4 hours. None of these three relapsed. All the 79 patients gave a widal reaction in dilutions of 1 : 100 or above.

By reviewing the dose schedules the following statistical summary is furnished by the authors, by selecting an arbitrary dose of 15 grammes of chloromycetin for patients in the first decade and 18 grammes for those in the second decade and above. Of 16 patients under 11 years of age, 14 were cured with 15 gms or less ; 2 relapsed after 12 gms.

Of 23 patients in the second decade (11 to 20 years of age) 18 were cured with 18 gms. or less ; 5 relapsed after 12 to 18 gms. Of 22 patients over 20 years of age 18 were cured with 18 gms. or less of chloromycetin ; 4 relapsed (3 after 12 gms. and 1 after 18 gms.)

Contrary to previous ideas held by the authors from earlier observations, there appears to be no correlation between length of time the person had been ill and the rate of response to therapy ; nor was there a correlation between age and response to therapy. No troublesome reactions to the drug were met with in these 79 cases. Those who relapsed gave a better response to second treatment with chloromycetin. Early and rapid disappearance of the toxic state was the first effect noticed following chloromycetin. An initial "*loading*" dose, advocated by some, was not used in the treatment of these 79 patients. The authors believe that such an initial heavy dose causes several "*antiphylactic*" reactions which have been noted. Massive and rapid death of bacteria in the body results in release of endotoxins. This explains the untoward reactions of antiphylaxis.

Clinical experience with P.A.S.—Preliminary report.—(*Dis. Chest*, Vol. 18, 5, pp. 413–429, Nov. 1950).

DeJanney, Cox and Grindell-Balchum of the Herman Keifer Hospital at Detroit, in a very exhaustive illustrated article, record their experience with 42 patients (40 with pulmonary tuberculosis and 2 with tuberculous meningitis) treated at their hospital with P.A.S. They state that the absorption of this drug is rapid and almost complete from the gastro-intestinal tract, and that it is rapidly excreted, 50% or more being removed by the kidneys. It is not found in urine 20 hours after discontinuing it. In one hour, urinary concentration reaches 400 mg. % level. Transitory urinary casts and transitory albuminuria have been observed but no permanent renal damage has so far been reported by any observer. The free acid has a rather unpleasant acrid taste, difficult to disguise and liable to cause mild gastric irritation. The sodium salt solution of this drug, though bitter, is not unpleasant, and the bitterness may be masked by flavouring agents. The material used was Pamisyl (P.D. & Co.'s P.A.S.) given in tablet form. Dose used was 9·6 to 10·5 gm. daily, administered five times, every 4 hours, during the day. 6 gm. was used to begin with and gradually increased to 9 or 10 gm. in seven days. Where clinical and X-ray improvement occurred and were

maintained the drug was continued for 90 to 120 days. The shortest was 30 and the longest 150 days. The age of the patients varied from 17 to 58, and 21 of these were in the 20 to 30 age group. Toxic manifestations noticed in this study were anorexia, nausea, diarrhœa, loss of weight and vomiting, all intermittent. There was no tinnitus vertigo and no evidence of hæmatopoietic renal or hepatic damage. Definite symptomatic improvement occurred in 22 of 37 patients who had any symptoms when P.A.S. was started. Some showed improved appetite instead of anorexia, and the symptomatic benefits consisted of improved sense of well being, reduction of fever, decrease in cough and sputum, and these occurred within one month of starting the treatment. Of 40 patients with pulmonary lesions 27 showed significant X-ray improvement while 13 showed no change. Six of these 27 had other treatment in addition to P.A.S.; the other 21 unquestionably represent a response to P A.S. Bronchoscopy showed subsidence of œdema, and reddening and healing of endobronchial ulcers in one month of P.A.S. treatment. Pulmonary tuberculosis which failed to respond to streptomycin, improved with P.A.S. in 67% of cases.

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Peripheral neuritis and bronchial carcinoma.—(Leading article, *Lancet*, 8-7-1950, p. 61).

To the many causes of peripheral neuritis must be added bronchial carcinoma. Denny Brown in 1948 (*J. Neurol. Neurosurg.*, 11, 73) described 2 cases in which these two disorders were associated. In each the neuritis was the presenting feature and the carcinoma was discovered only post-mortem. Wyburn-Mason described (*Lancet*, 1948, 1, 203) the same 2 cases and added a third in which the clinical involvement was motor rather than sensory. Lennox and Prichard have now (*Quart. J. Med.*, 1950, 19, 97) described two further cases of this strange association. Clinically motor elements of the peripheral nerves were mostly affected ; at necropsy demyelination of motor fibres was observed. These authors further found records of three further cases of bronchial carcinoma associated with peripheral neuritis and the symptoms in these three were neurological. Denny Brown impressed by the unusual clinical and pathological features of the peripheral nerve disorders concluded that the two diseases were intimately connected ; Lennox and Prichard's cases were five among 299 of bronchial carcinoma ; that the association is not fortuitous is strongly suggested by their review of the incidence of peripheral neuritis in cases of carcinoma at other sites and of other types of chronic chest disease. This association of diseases indicates the importance of radiography of the chest in cases of peripheral neuritis of obscure origin ; and it will be interesting to study the course of the neuritis if a case is found in which the bronchial carcinoma can be removed completely.

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Medical management of acute lung abscess.—(Jewett, J. S. and Dimond, G. E., *Dis. Chest*, 18, 5, pp. 478-500, Nov. 1950).

During 2 years, the authors have observed 28 cases of lung abscess, 11 of these with acute suppuration in a previously healthy lung and one with an acute abscess in a bronchiectatic lobe. The management of acute uncomplicated lung abscess has been greatly modified in the last few years. The pendulum has swung away from early pneumonotomy in favour of antibiotic therapy. It has been recorded (*J. Thorac. Surg.*, 18, 618, 1949) that surgical interference is generally of no value in the management of acute, uncomplicated nonputrid lung abscess and that in many cases it may be absolutely harmful. Six factors have to be taken into account in deciding on the therapy : (1) Cause of abscess ; (2) duration of abscess ; (3) condition of patient ; (4) nature and virulence of infecting organism ; (5) site and extent of lesion ; and (6) presence of complications in the lung of pleura.

Of the 12 cases all but one were 'primary'. Eleven responded to parenteral aerosol penicillin, In 10 of these, radiographic cure resulted. Penicillin therapy offers an entirely satisfactory means of treating acute uncomplicated lung abscess. Early pneumonotomy is no longer necessary

as long as an abscess remains uncomplicated. Penicillin treatment should be continued until complete healing has been demonstrated by rœntgenologic analysis. The authors conclude by saying that their experience has impressed the necessity for a thorough investigation of every case of lung abscess for bronchogenic carcinoma, as well as bronchiectasis.

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Infarction of the heart.—The role played by cholesterol in diet.—(Malmros, H., *Report to Swedish Med. Soc.*).

Figures quoted by Dr. Haqiru Malmros of the Orebro Central Hospital, Sweden, tend to substantiate the claim that coronary thrombosis with infarction of the heart is fast becoming a common ailment. He found that there were only 23 deaths from this cause per 1,00,000 in Norway in 1945, as compared with 150 such deaths in Sweden per 1,00,000. In other words, the death rate from this cause was more than 6 times higher in Sweden than in Norway.

These and various other challenging observations have led Dr. Malmros to seek explanations in the differences in the diets of the 2 nations. He wonders whether diets rich in cholesterol could be responsible for the arteriosclerosis and infarction of the heart. He questions the current overproduction of eggs in Sweden; could this be a threat to the health of the nation, at least that section which is predisposed to xanthematosis or Muller's disease? The 50 million eggs in storage in Sweden at one time in 1949, may perhaps be a *national danger*, rather than a national asset, and Dr. Malmros suggests that if the problem cannot be solved by exporting the surplus of eggs it would be well to kill off many of the hens producing them. The Swede who is predisposed to this class of disease would also do well to ration himself with regard to butter, eggs and other foods rich in cholesterol. Evidently there are many in this class, as judged by the 27 cases of xanthelasma of the eyelids and arcus corneæ discovered by Dr. Malmros during a fortnight's survey of the occupants of the medical department of his hospital.

NEWS AND NOTES

Key Factor in Life Process (chemical) Isolated in U.S.A.—An American scientist has isolated a chemical compound that appears to be vital in the fundamental mechanisms by which plants and animals live and grow.

Called triphospho-pyridine-neuclotide (TPN), the compound is believed to be a factor in photosynthesis—the process by which plants synthesize sugar and other foods from water and carbon dioxide by using the energy of sunlight. Enzymes, or natural catalysts, guide the process. In the animal, conversely, TPN may aid in changing digested foods into water and carbon dioxide with the release of energy.

During photosynthesis, TPN appears to work by temporarily "holding" hydrogen after water has been split into oxygen and hydrogen by chlorophyll, the green colouring matter in plants. With the highly reactive hydrogen removed, oxygen enters into the building of complex food substances.

When plants use their own stored food or when animals eat plant foods, TPN again appears to play the role of hydrogen acceptor as food is broken down into water and carbon dioxide.

In recent experiments, scientists placed animal TPN in a flask with enzymes from pigeon livers and added chlorophyll from spinach and carbon dioxide. Under a strong light a complex sugar was produced, indicating that the process similar to the one that occurs in natural life had been duplicated.

For a long time TPN has been known to be a component of plant and animal tissues. Its purpose, however, had not been known. The recent experiments were conducted by Dr. Severo Ochoa of the New York University Medical School.—*U.S.I.S. Extract.*

* * *

New Heart Drug.—Tromexan, a new drug, has been found in recent tests to be the most effective agent used to prevent clotting of the blood. It is quicker and gives fewer apparent side-effects than any other anti-coagulant used to date, the American Heart Association reports.

At the Los Alamos Atomic Energy Project in New Mexico, the action of Tromexan was compared with that of dicumarol, another anti-coagulant. Both drugs were treated with radioactive carbon 14 so that their absorption, metabolism and excretion could be observed in laboratory animals. Scientists found that Tromexan was used more rapidly and with less prolonged effects, decreasing the danger of hæmorrhage.

At the Cornell College of Medicine at Ithaca, New York, Tromexon later was used successfully with 112 human patients who had thrombosis—the clotting or blocking of a blood vessel. Studies with anti-coagulants are a major part of the stepped-up

attack by the American Heart Association on heart disease.—*U.S.I.S. Extract.*

* * *

Cancer clues sought.—American doctors are investigating the personal habits and working condition of cancer patients to learn more about the factors that may contribute to cancer.

Lung cancer patients in the State of Massachusetts will be asked about their smoking habits, previous lung diseases, family medical history and exposure to occupational hazards. In particular, doctors hope to determine how much the smoking of tobacco influences the development of lung cancers.

How working materials such as nonferrous metals are related to cancer development will be investigated in hospitals in the State of Connecticut. Doctors will collect job histories on all types of cancer patients and then compare these findings to studies of suspected hazards to which the workers were exposed. The United States Public Health Service recently made grants for these studies.

* * *

Examination in Indian Medicine.—The Government of Madras have revised the rules for the G. C. I. M. and H. P. I. M. examinations at the College of Indigenous Medicine. They have directed that 20% of the marks in practical examination of the G. C. I. M. course in anatomy, physiology, pharmacology, hygiene and pathology be allotted to the practical work done in the classes and that these marks be taken into account at the time of the Government examinations. The marks will be awarded by the Government Examiners to whom the class notes should be submitted for scrutiny.

The Government have approved the proposal of the Board of Examiners in Indigenous Medicine for the revision of rules regarding the conduct of examination for the Diploma of High Proficiency in Indian Medicine (H. P. I. M.). Candidates who have already appeared under the existing regulations, and have failed in either of the groups will continue to be governed by old regulations, while candidates who have failed in both groups under the old regulations will appear for the examination under the revised curriculum with effect from April 1951.

* * *

WHO to award more than 250 Fellowships this year.—Fellowships for advanced study will be awarded this year by the World Health Organisation to more than 250 persons in medicine and allied fields throughout the world.

Those eligible are technical and teaching personnel from WHO member States with at least two years' experience in the fields in which the fellowships are to be awarded. Applicants must apply through their Governments.

Approximately 800 fellowships already have been awarded by WHO in a variety of fields. These include, for example, public health administration, communicable diseases, nursing, maternal and child-health and surgery.—*U.S.I.S. Extract.*

* * *

Compulsory service in villages.—The Government of Bombay were considering a proposal to send the new medical graduates compulsorily to villages to serve the people, Dr. M. D. D. Gilder, Minister for Health, told the State Assembly on March 16, in reply to a cut motion to discuss the Government's failure to give adequate medical relief to the people in rural areas. He also said that the Government were faced with the greatest problem of finding medical personnel willing to go to villages. They were also experiencing shortage of trained medical personnel and non-availability of some drugs.

* * *

Machine for talking to the Deaf-Blind.—To make contact with the deaf-blind a knowledge of Braille has always been essential. Now, thanks to a brilliant new invention, it will be possible in future for anybody to hold conversations with them. A. R. Cooper, an Electrical Engineer, with the help of other electrical experts, worked out the theory of a machine which would enable anybody to talk to the deaf-blind. Then the theory was put into practice and the machine constructed. At one end of the machine is a type-writer keyboard, with the letters arranged in alphabetical order to make it simpler for people with no knowledge of typing to operate it. At the other end are six apertures over which the deaf-blind person places his hands. As the type-writer keys are depressed, plungers strike the fingers conveying the Braille characters. They are actuated by a system of electro-magnets. Mr. George, Robinson, Superintendent of the Liverpool Adult Deaf and Dumb Institute, to whom the machine was shown said, that here was something which could make a world of difference to the lives of the deaf-blind. He says: "I think it is a marvellous invention. It will supersede the existing practice of talking by touch on the person's hands. The very novelty of the machine will encourage people to use it—people who would never normally learn Braille and could, therefore, never talk to the deaf-blind". The next step is the construction of more machines and a large scale test to decide whether any modifications are necessary.—*B.I.S. Extract.*

* * *

Industrial Health Services.—The Committee of Inquiry appointed by the British Prime Minister, Mr. Atlee, to examine the relationship between these services and the general preventive and curative health services provided for the general population has just issued its report. The report bears emphatic testimony to the essential importance of the industrial health services, from whatever angle they are regarded and treats them as a normal function of enlightened management to-day. The services are of great value because of the contribution they

make, says the Report, both to the health of the workers and to productivity. By taking care of his health, these services help to keep the worker at his employment, contribute to his confidence and thus increase his satisfaction with his work and conditions. "They are thus an important element in promoting and maintaining good morale in industry," says the Report.

One of the recommendations of the Committee, on which two representatives of the trade union movement served is, that a new permanent advisory council of doctors, nurses, workers and employers should be set up along with the representatives of the Ministries of Health, Fuel and Power, and Labour, to act as a link between the national public, and industrial health services.—*B.I.S. Extract.*

* * *

More Women want to be Doctors.—It is estimated that there are about 8,000 women doctors in Britain who are qualified to practise the calling of a physician. Each year more and more young women students enter the various medical schools attached to Universities and hospitals in England and Scotland—2931 in 1950 as compared with 1,930 in the year immediately before the outbreak of World War II.

* * *

Trials with PAS yield striking results.—Hitherto it has been the policy not to give streptomycin to patients who can reasonably be expected to recover merely by prolonged rest, on the ground that it may breed resistant bacilli which could not be attacked by the drug if a relapse occurred later. PAS, the results of the trial of which have been striking, seems capable of reducing this risk so much that it may prove practicable to use streptomycin in a much earlier stage in pulmonary tuberculosis than is now customary. The appearance of some drug-resistant bacilli at a late stage in treatment need not necessarily make this unsuccessful, for if the degree of infection can be substantially reduced the body's own natural defences may well prove capable of dealing with the residue which the drug will not attack.—Dr. Trevor, I. Williams, Deputy Editor of the Scientific Journal "*Endeavour*"; *B I.S Extract.*

* * *

Production of PAS in India.—Replying to an interpellation in the Indian Parliament on April 10, 1951, the Health Minister stated that a former research scholar of the University College of Science of Calcutta had evolved a method for the technical production of PAS, a specific for tuberculosis. The doctor was working with a firm now producing PAS.

* * *

Madras Medical Service—Single Cadre of Assistant Surgeons formed.—The Government of Madras have passed orders, unifying Classes I and II of Civil Assistant Surgeons in the Madras Medical Service into a single cadre of Assistant Surgeons.

The Order states that as there has been no licentiate course in Medicine in this State from about 1939, only medical graduates will be available for appointment to the posts of Civil Assistant Surgeons, Class II. It has also been found that the scales of pay of Civil Assistant Surgeons Class II, are not sufficiently attractive for graduates, and that, in view of the large number of posts in Classes I and II, much administrative inconvenience is caused. The Government have, therefore, decided that the cadres of Civil Assistant Surgeons, Classes I and II, shall be unified to form a single cadre of Assistant Surgeons with effect from April 1, 1951, on the scale of pay of Rs. 150—25/2—400, medical graduates being allowed to start on Rs. 200/- in the scale.

The pay of every officer holding a post in the cadre of Civil Assistant Surgeons Class II, at the time of the unification of the cadre of Civil Assitant Surgeons, Classes I and II, shall be fixed in the scale of Rs. 150—25/2—400 at the stage next above his pay in the old scale of pay, subject to the exercise of the option allowed under Fundamental Rule 23. This option should be exercised on or before March 31, 1952, failing which their pay will be fixed in the new scale of pay with effect from April 1, 1952. Medical graduates, drawing pay of less than Rs. 200 per month, shall be eligible to draw Rs. 200 in the new scale of pay from April 1, 1951. Officers who hold appointments in this cadre of Civil Assistant Surgeons, Class I, at the time of the unification shall continue to draw the pay they are drawing in the existing scale of Rs. 200—25/2—400.

Assistant Surgeons, whether licentiates or graduates, who have been entirely debarred from engaging themselves in private practice, shall be allowed to draw a compensatory allowance of Rs. 80/- per mensem. Assistant Surgeons, whether licentiates or graduates, whose private practice has been restricted to consulting practice, shall be allowed to draw a compensatory allowance of Rs. 50/— per mensem.

* * *

The Organising Secretary, Inaugural Meeting of the Anatomical Society of India, Department of Anatomy, Nilratan Sircar Medical College, Calcutta writes :—

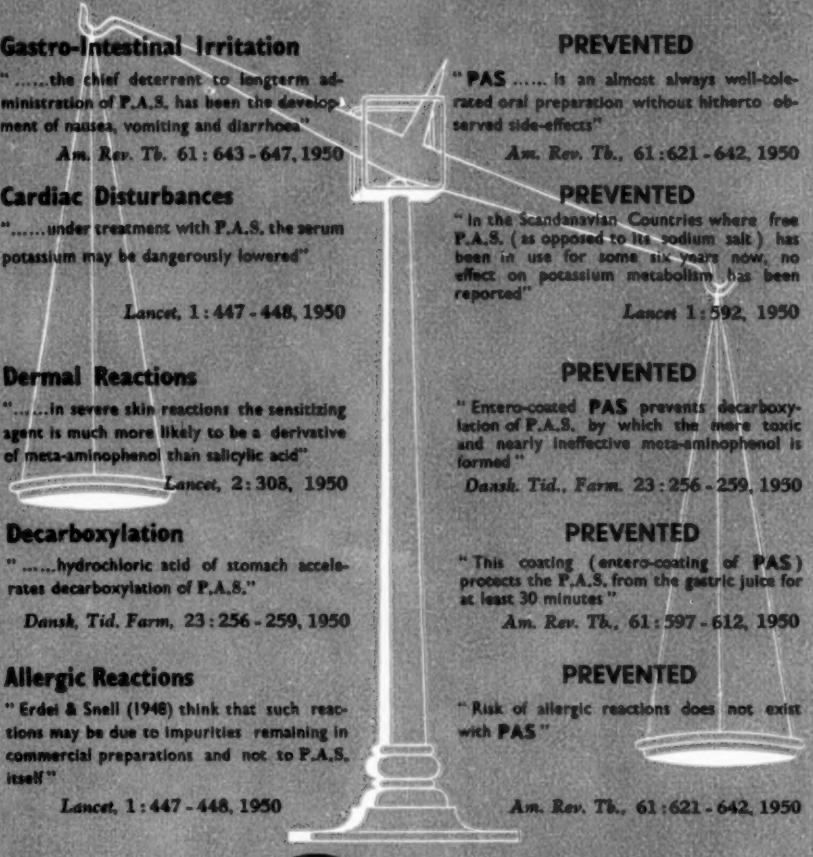
The Inauguration Ceremony of the Anatomical Society of India will be held on the 20th May, 1951 at 9.30 a.m. at the Medical College of Bengal, Calcutta.

Dr. M. N. Bose, Principal, R. G. Kar Medical College, Calcutta, will preside over the function and the Hon'ble Dr. B. C. Roy, Chief Minister of the State of West Bengal will formally inaugurate the Society.

On behalf of the persons interested in the subject in India the Secretary extends to you a very cordial invitation to attend the function.

In case it may not be possible for you to be present, he requests you to send your blessings and message of goodwill.

Weigh these Facts –



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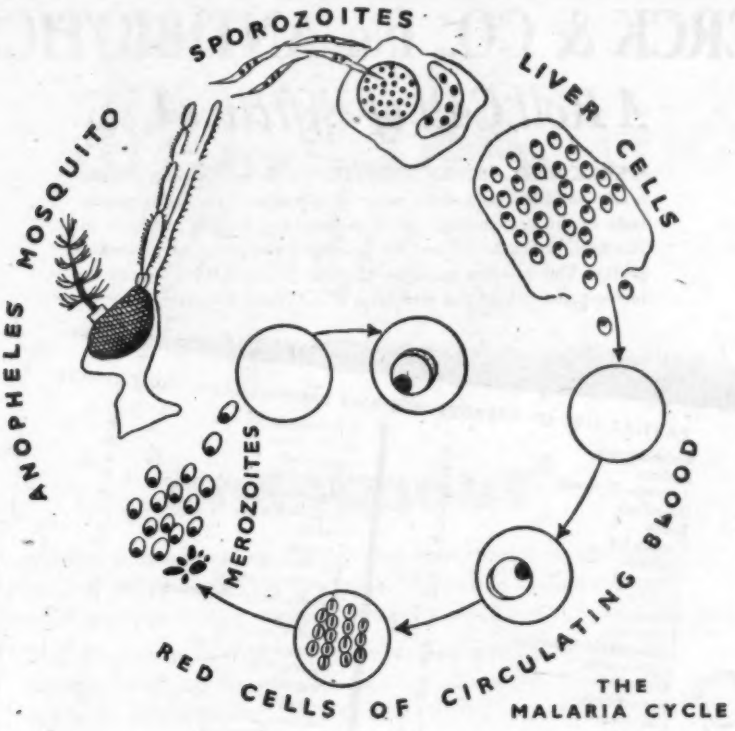

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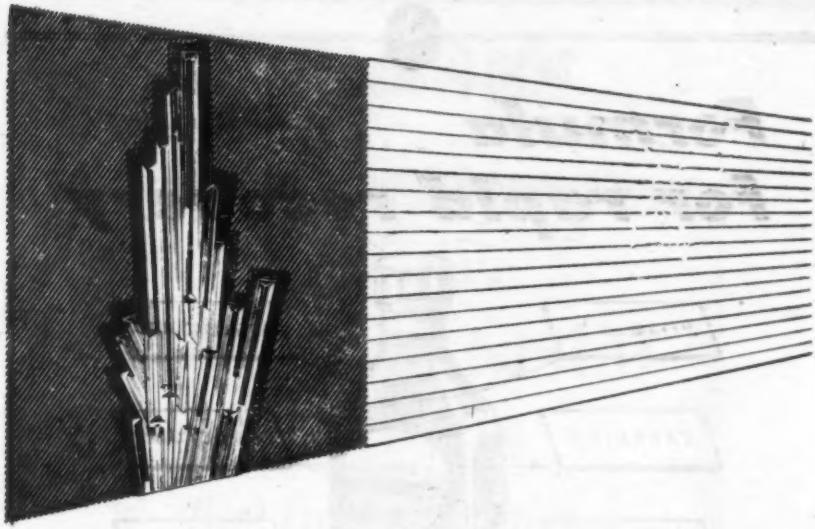

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